ADHERENT PERICARDIUM

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FREQUENCY OF OCCURRENCE OF ADHERENT PERICARDIUM AS SHOWN BY POST-MORTEM STATISTICS—EXPLANATION OF ITS FREQUENT OCCURRENCE—HISTORY AND LITERATURE OF THE SUBJECT.

By the term 'adherent pericardium' is implied the existence of adhesions between the visceral and parietal layers of pericardium. These may be limited to fibrous bands stretching across the pericardial cavity; or they may be universal, in which case the pericardium and heart are so intimately connected that the pericardial cavity is entirely obliterated. (It is this latter condition that is implied when the term "adherent pericardium" is used in this treatise.) Adhesions may also exist between the chest wall and pericardium as well as between the two layers of pericardium, as a result of so-called mediastino-pericarditis.

The number of cases of adherent pericardium seen in the post-mortem room, and the comparatively few cases in which a diagnosis of this condition is made before death, make this subject worthy of special study and investigation.

On consulting the post-mortem records of St. Mary's Hospital for the years 1890–93, I noted that, out of eighty-six cases in which death was attributable to heart disease, in no fewer than thirty-one was adherent pericardium found at the autopsy—in the majority of cases, viz. twenty-one, as a complication of valvular disease.

Nor is it remarkable that we should find pericardial adhesions in so large a proportion of cases of morbus cordis in adults when we turn to the post-mortem statistics of a Children's Hospital. Dr. Sturges pointed out * that pericarditis is very common in children, and may occur alone, or as a complication of endocarditis, but that endocarditis unaccompanied by pericarditis is comparatively rare in childhood. This statement he verifies by statistics, as follows :—

Out of one hundred fatal cases of heart disease occurring at the Children's Hospital, Great Ormond Street, between June, 1881, and April, 1892, of which fifty-four were of rheumatic origin, and forty-six due to other causes, in six only was there no evidence of pericarditis. It is not, therefore, to be wondered at that we should find traces of old pericarditis, in the shape of pericardial adhesions, in many of those who suffered from affections of the heart during childhood.

* *Lancet*, Aug. 27, 1892, and again in the 'Lumleian Lectures,' 1894.

The comparative rarity with which the existence of adherent pericardium is diagnosed may be accounted for in many instances by the fact that it is not thought of. Especially is this the case when it is associated with valvular disease, for the valvular lesion is judged to be sufficient to account for the symptoms that arise. In many cases there may be no characteristic physical signs or symptoms to denote its existence, and when unaccompanied by valvular disease there may be no special indications to point to the heart as the source of the trouble.

Hence the diagnosis is frequently a matter of considerable difficulty.

History and Literature of the Subject.

Galen, and most of the early writers, believed that in cases of adherent pericardium there was a congenital absence of the pericardial sac.

Gentilis of Fuligno, 1518, attributed this condition to hypertrophy of the pericardium, accompanied by atrophy of the heart.

Boerhave, in his writings collected and published by Haller, mentions adherent pericardium as a cause of palpitation and cardiac distress leading subsequently to death. The adherence of the pericardium he attributes to deficiency of 'vapor' in the pericardial cavity. After stating that all the cavities in the body normally contain this 'vapor,' the absence of which occasions serious disease, he ends up with the following words: 'Eo enim vaporo deficiente,

visum est cor cum pericardio connatum post miseras angustias et palpitationes, homines occidisse.' *

About the end of the seventeenth and the beginning of the eighteenth century, Lower, Vieussens, and Lancisi began to investigate the subject from a clinical and diagnostic point of view.

Vieussens describes a case of adherent pericardium in a child of five, and mentions palpitation and pain in the præcordium among the symptoms, also dyspnœa, which he attributes to the inability of the diaphragm to descend perfectly in inspiration, owing to its being bound down to the heart by pericardial adhesions.

In a case which he quotes the pericardium was found to be thick and firm, and almost cartilaginous at the autopsy. He makes a great point of the hampering of the movements of the diaphragm by the adherent pericardium, and describes it in the following words: 'Toutes les fois que le péricarde se colle au cœur il se raccourcit, et parce qu'il est attaché à la partie tendineuse du diaphragm, il le porte vers le dedans de la poitrine et l'empêche de s'applanir librement dans l'inspiration.' †

Lower describes a case of adherent pericardium which he attributes to deficiency of fluid in the pericardial cavity. He gives as prominent symptoms in such cases, general anxiety and depression of spirits, pain in the præcordium; shortness of breath on exertion. He considers the irregularity of pulse,

* 'Prælectiones Hermani Boerhave,' edited by Haller, 1740.
† Vieussens, 'Traité des Mouvements, etc., du Cœur,' 1715, ch. i. pp. 15, 16.

which he also mentions as occurring in the case, as due to the fact that the descent of the diaphragm in inspiration draws down and pulls on the heart, and so interferes with its normal movements.*

Lancisi gives an account of a case of adherent pericardium with autopsy. The chief symptoms were: small and irregular pulse, dyspnœa; fulness of the jugular veins; pain in the præcordium; general *malaise* and loss of appetite. He comments on the case, and explains how the pericardium may have become adherent, at the same time emphasizing the fact that such a condition is not due to a congenital absence of pericardium: 'Mihi enim satis est innuere, languescenti motu cordis, deficiente intermedio liquido, pericardium minimo negotio cordi adhœsisse, et cum eodem magis magisque agglutinari cepisse, ut erroris causam patefaciat illorum qui cor interdum pericardio destitutum se reperisse affirmunt.' †

Morgagni discusses very fully the question of adherent pericardium. He states that many writers had mistaken cases of adherent pericardium for absence of pericardium, and refutes this idea. He gives an account of forty-five cases of adherent pericardium, collected from the writings of various authors, whose names he gives, and he describes the main features of each case. He states that palpitation had been described as a prominent symptom of adherent pericardium by Vieussens and other writers, but this he does not regard as a characteristic symptom: it was present in only fifteen out of the forty-five cases described.

* Lowerus, 'Tractatus de Corde,' 1727, ch. ii. p. 108.
† Lancisi, 'De Mot. Cordis,' 1728, propositum xxiii.

Absence of or weakness of the apex beat he considers an important physical sign; from an analysis of these cases, finding it thirty times. He describes three cases that Valsalva had had under his observation, and seven cases of his own with autopsies. He gives a short clinical account of each case with the autopsy, and follows it up with a commentary. He mentions smallness and irregularity of pulse, palpitation, shortness of breath, œdema, and ascites as some of the symptoms, but in one of his cases there were no characteristic features by which a diagnosis of adherent pericardium could be made. He admits that the diagnosis is in most cases very difficult.

He mentions that Vieussens and Lower have noted dyspnœa, irregularity of pulse, and pain in the præcordium as occurring in cases of adherent pericardium, and he explains their cause in the following clear and instructive manner.

With regard to the dyspnœa, he says: 'Nonne ob hanc pericardium contractius factum, annexum sibi a natura diaphragma retractum servat, ejusque, cum spiritus ducitur, depressionem minus facilem reddit, idque eo magis quo pericardium durius est?' With regard to the irregular pulse, he continues: 'Nonne descendens tunc diaphragma pericardium secum rapiendo efficit ut magis a pericardio cor constringatur, et sic multo difficilius cavearum expansio fiat, unde oppressio, pulsus intermissio et palpitatio consequatur.' *

The references to the cases of his own which he describes are ep. iv. n. 19; ep. v. n. 19; ep. viii. n. 6;

* Morgagni, tom. iii. lib. ii. epist. xxiii. n. 22, p. 244.

ep. xviii. n. 25; ep. xxiv. n. 11; ep. xxx. n. 7; ep. xxxiv. n. 12.

Lieutaud gives an account of twenty-three cases of adherent pericardium, with the chief symptoms and with autopsies, but he makes no comments on them. The chief symptoms mentioned are dyspnœa, anxiety, cough, small and irregular pulse, œdema, and ascites.*

Corvisart states that adherent pericardium is necessarily accompanied by disturbance of the functions of the heart, and ultimately proves fatal.

He describes three cases of adherent pericardium which he had seen, in the first of which palpitation, small and irregular pulse, dyspnœa, and pain in the præcordium were the chief symptoms. In each case the patient improved at first, but subsequently died eight months after first appearance of symptoms with general œdema and ascites. In the third case he mentions intense mental depression as a prominent feature, which ultimately caused the patient to commit suicide by taking opium.

He, like Morgagni and Vieussens, lays stress on the part which the adherence of the heart to the diaphragm, through the pericardial adhesions, plays in the production of symptoms, especially mentioning the præcordial pain: 'Le malade éprouve aussi un sentiment pénible de tiraillement dans la région du cœur, parce que dans l'acte de respiration, le diaphragm entraine dans son abaissement le péricarde et tout le cœur qui lui est devenu adhérent et s'oppose dans ce temps de son action au mouvement particulier

* Lieutaud, 1767, 'History Anatom. Medic.,' tom. ii. lib. ii. p. 72, sqq.

d'élévation propre du cœur lors de ses contractions.' * He regards the absence of a marked thrill as an aid to diagnosis, as indicating that the heart is so tied down by adhesions that it cannot execute movements of any amplitude. He says that the diagnosis is difficult, especially if there are also complications in the shape of other diseases of the heart or lungs.

Heim, according to Kreysig, made an important observation with regard to the diagnosis of adherent pericardium. He noted that in some cases there was marked retraction during the cardiac systole of the epigastric region to the left of the sternum and beneath the false ribs.†

Aran, in a paper on adherent pericardium, says he believes he has discovered a sign of great diagnostic value, not mentioned by any previous writer on the subject, namely, 'the feebleness and extinction more or less complete of the second sound of the heart, both at the apex and base.'

He quotes three cases in support of this statement. The second case he describes was complicated by mitral stenosis, and he does not appear to take into account the fact that this mitral lesion would in itself tend to cause feebleness of the aortic second sound. It is difficult to understand how he came to his conclusions. His paper is to be found in 'Arch. Gen. de Medicine,' tom. iv. p. 476.

Hope agrees with Vieussens and Corvisart that a patient with adherent pericardium cannot live in a state of health. He makes a great point of hypertrophy

* Corvisart, 'Maladies du Cœur,' 1818, p. 37.
† Kreysig, 'Die Krank des Herz,' ii. 625.

of the heart, which, he says, must take place in all cases of adherent pericardium, and states that in all the autopsics he has made he has invariably found considerable enlargement of the heart. He explains why such hypertrophy should take place in cases of adherent pericardium, in the following words: 'How adhesion occasions hypertrophy, may be easily understood: firstly, inflammation is probably a cause of hypertrophy; secondly, the organ must increase its contractile energy to contend against the obstacle which the adhesion shackling its movements presents to the due discharge of its function; and increased action leads to increase of nutrition as explained in the article on hypertrophy.' *

He mentions two signs which he regards as important aids to diagnosis. To quote his words—

'The heart, though enlarged, and when, therefore, it ought to beat preternaturally low in the chest, beats as high up as natural, and sometimes occasions a prominence of the left præcordial ribs.'

The second sign he describes as follows:—

'An abrupt jogging or tumbling motion of the heart, very perceptible in the præcordial region with the cylinder [wooden stethoscope]. It is more distinct when the heart is hypertrophous and dilated. Under these circumstances I have found the jogs correspond with the ventricular systole and diastole respectively, that of diastole being sometimes nearly as strong as the other, and bearing the character of a receding motion suddenly arrested.' †

* Hope, 'Diseases of Heart,' 1839, p. 192.
† Ibid. 1839, p. 195.

Williams, 1840, noted that when the pericardium is adherent both to the heart when enlarged, and to the walls of the chest, the heart pulsates in close contact with these walls, so that the pulsations are felt widely extending upwards as well as downwards, and drawing in the intercostal spaces during systole. He also pointed out that in these conditions the area of cardiac dulness remains unchanged during expiration and inspiration, and that the position of the apex beat remains unaltered in all postures of the body; further, that the pericardium and heart, being fixed to the diaphragm and also to the chest wall, the descent of the diaphragm in inspiration on the left side is interfered with—a fact which had already been noted by Vieussens and Morgagni.*

In his edition of 'Diseases of the Chest,' 1835, Williams remarks that, 'when the heart is enlarged and adherent by its upper parts, it will pulsate with increased force, but not lower down than natural, as in simple enlargement; but if the adhesion is lower down or loose, this result will not be observed.'

Skoda made a special study of adherent pericardium, and in 1852 published an important paper on the subject. He lays great stress on the diagnostic importance of recession during the cardiac systole of the intercostal spaces to the left of the sternum, which phenomenon he believes can only occur in cases of adherent pericardium, the spaces being drawn in by the contraction of the adherent heart.

He also notes the diastolic shock which immediately follows the retraction, due to the elastic recoil of the

* Williams, 1840, 'Diseases of the Chest.'

chest walls. This can only occur when the pericardium is adherent both to the heart and to the anterior chest wall.

After giving a critical account of several cases of adherent pericardium described by different authors, he describes three cases observed by himself. In two of these he noted systolic retraction of the third, fourth, and fifth left intercostal spaces, with no alteration of the area of cardiac dulness in inspiration and expiration; in two, the apex beat was imperceptible. In one there was considerable retraction of the fifth space during systole, followed by a diastolic shock. In all three the right ventricle was considerably enlarged. The paper with the account of the cases is to be found in 'Zeitschrift der Gesellschaft der Aerzte zu Wien,' 152. 1, 306.

Gairdner, in 1851, wrote a paper 'on the favourable terminations of pericarditis' in the *Edinburgh Monthly Journal of Medicine*. He claims that adherence of the pericardium does not necessarily prove rapidly fatal, but that a person may enjoy good health with a universally adherent pericardium, though, if the patient suffer from some intercurrent disease, he is more likely to succumb to it.

Kennedy, in 1858, wrote a paper on 'Adherent Pericardium and its Results,' giving a number of cases, but he does not throw much additional light on the subject.

He gives an account of ninety cases of adherent pericardium uncomplicated by valvular disease, with a view to showing in what proportion of cases hypertrophy or atrophy of the heart results, and in what

number the heart remains apparently normal in size. In thirty-four cases out of the ninety he finds that the heart remains unaltered; in fifty-one it undergoes hypertrophy and dilatation; in the remaining five it appears to be atrophied.*

Gairdner follows up this paper of Kennedy's with another in the next number of the *Edin. Med. Journal.*

He states that he believes Kennedy's conclusions are unreliable, as many of the cases seem to be selected from Museum catalogues, and it is probable therefore that the proportion of hearts markedly hypertrophied is too great, as they would be more likely to be kept as museum specimens than hearts of normal size.

He says that out of five hundred cases examined by himself post-mortem there were only fifteen cases of adherent pericardium. In ten of these the condition of the heart was healthy, *i.e.* there was no dilatation or hypertrophy; in five only it was morbid. In two out of these five valvular disease was present as well. He claims, therefore, that enlargement of the heart as a result of adherent pericardium is not nearly so common as Kennedy's statistics would make it—viz. 56 per cent.; as from his own cases the proportion would only be about 33 per cent. On these grounds he maintains that the prognosis in cases of adherent pericardium is, in the majority of cases, fairly good.

Friedreich described peculiar phenomena pertaining to the cervical veins in cases of adherent pericardium; these consist in sudden diastolic collapse of the cervical veins and frequently deepening of the supra-clavicular fossæ at the same time.

* Kennedy, *Edin. Med. Journal*, May, 1858.

These phenomena, he says, are connected with the systolic retractions of the intercostal spaces, and are not present without them; this diastolic retraction, according to Friedreich, is not to be confounded with a venous pulse.

With regard to the systolic retractions of the intercostal spaces to the left of the sternum, regarded as pathognomonic by Skoda, he has shown that they may occur in cases where there are no pericardial adhesions; this Traube has also confirmed. He has also noted that a diastolic shock may be felt in some cases where there is systolic retraction of the area of the apex beat; he further states, that in such cases the rebound of the chest wall produces a dull sound which may be heard on auscultation after the second sound of the heart; in this way the second sound seems split or doubled.*

Sibson gives a short history of recent writers on adherent pericardium, and discusses fully the various physical signs and symptoms described by them; he further gives a full and very instructive account of several cases of adherent pericardium carefully observed by himself.†

Wilks holds the view, that in persons of mature age, where severe heart symptoms are present without valvular murmurs, degeneration of the cardiac muscle must be inferred, but that in the young the same state of things points to pericardial adhesions.

* N. Friedreich, 'Zur Diagnose der Herzbeutel Verwachsung,' Virchow Arch. 29 Bd. 1864.

† Reynolds, 'System of Med.,' vol. iv. p. 438, or vol. iv. of 'Sibson's Works,' ed. by Ord.

CHAPTER II.

ETIOLOGY OF ADHERENT PERICARDIUM—MORBID ANATOMY —EFFECTS ON THE HEART—PHYSICAL SIGNS.

Etiology of Adherent Pericardium.

Every adhesion of the layers of the pericardium is the result of antecedent pericarditis. A single acute attack of pericarditis does not necessarily give rise to pericardial adhesions unless it is protracted and assumes a chronic form; they are more liable to result from repeated attacks of pericarditis which are from the outset subacute and are of long duration. Partial adhesions may take place during one attack and become universal during the next. At an autopsy, firm adhesions of old standing may be found, side by side with easily broken down recent adhesions, the result of the attack which has proved fatal. In the rheumatic pericarditis of children of a subacute nature there is usually little effusion into the pericardial cavity, but an exudation of a thick, sticky lymph, which coats both layers of the pericardium and gives a honeycomb or bread-and-butter-like appearance when separated. It is easy to understand

how this lymph may become organized into fibrous tissue firmly uniting together the two layers of pericardium.

Morbid Anatomy.

The two layers of pericardium may be universally adherent to one another, so that the pericardial cavity is entirely obliterated. The adhesions, if of old standing, are tough and fibrous, so that the pericardium cannot be stripped off without tearing the heart substance.

Partial adhesions may exist in the form of fibrous bands, stretching between the two layers of pericardium across a pericardial cavity. The pericardium may also be adherent to the chest wall or adjoining pleura and lung substance. There may be adhesions, too, between the pericardium and the œsophagus, aorta, and vertebral column. The fibrous tissue binding together the two layers of pericardium may undergo calcareous degeneration, so that irregular hard plates of calcareous matter may be found in cases of very old standing. Where the adhesions are recent, we may be able to separate the two layers; and occasionally remains of imprisoned exudation, pus, or cheesy substance are found between them.

Effects of Adherent Pericardium on the Heart.

The heart itself may be enlarged and hypertrophied, or atrophied, or normal in size. Frequently the heart

is dilated and hypertrophied, but hypertrophy is by no means a neeessary consequenee of adherent pericardium as Hope believed. Tho heart may be of normal, or sometimes of lcss than tho normal size. When thc heart is apparently atrophied, the pericardial adhesions are usually dense and firm, and appear to have, so to speak, strangled and eompressed the heart so as effectually to prevent onlargement of it, or even its normal development, in thc ease of a child who arrives at maturity. It is more diffieult to aeeount for the enlargement of the heart that is often found in assoeiation with, and is apparently due to, adhorent perieardium.

Hope went so far as to state that adherent pericardium is always aecompanied by cardiac hypertrophy, and attributed the hypertrophy to the extra work thrown on the heart by the hampering of its movements by the adhesions. This statement is not borne out by faets, as Kennedy and Gairdner have pointed out; nor can Hope's explanation hold good, for, were it true, we ought to find the heart hypertrophied in all cascs of adherent pericardium, which is not the ease. Why, then, is the heart normal in size in some eases, and eonsiderably enlarged in other cases of adherent pericardium? The explanation I would suggest is the following: When the heart is found to be dilated and hypertrophied as a result of adherent perieardium, there being no valvular disease to aeeount for it, it is due to the fact that it has been left in a condition of dilatation after tho original attaek of pericarditis, and that whilc in this condition of dilatation the pericardium has becomo adhcrent;

then the adhesions becoming organized, the heart is effectually prevented from again recovering its normal size. Subsequently it undergoes some hypertrophy.

The following are the steps in the process: During the attack of pericarditis the heart becomes considerably dilated in consequence of the myocarditis accompanying it; after the subsidence of the attack the heart remains enlarged. It is obvious that the fact of the heart being enlarged and dilated would favour the formation of adhesions by the approximation of the walls of the heart and the pericardium. A further contributory cause will be the fact that the heart, in a condition of dilatation, beats with less force and has less power to free itself from adhesions when they form. Again, as the cardiac dilatation is due to myocarditis accompanying the pericarditis, the contractile power of the heart must be impaired, not only at the period of the attack, but for some time after, so that it remains dilated till the pericardial adhesions have become organized into firm unyielding fibrous tissue, which prevents it again contracting down to its normal size.

This would explain why the heart should in some cases of adherent pericardium be found considerably dilated and hypertrophied.

The explanation why, in other cases, the heart should be of normal size would be that it had not dilated during the original attack of pericarditis, or else had recovered from its dilatation before adhesions were formed.

Later on, in the discussion of physical signs of adherent pericardium, cases are related proving (1)

the existence of marked cardiac dilatation during an attack of pericarditis; (2) the association of adherent pericardium with this condition of dilatation in cases where it remains permanent, and is unexplained by valvular disease or other cause.

Physical Signs of Adherent Pericardium.

The physical signs differ according as the adhesions exist only between the two layers of pericardium or between the pericardium and chest wall, or adjoining pleura, as well. In the latter case they are more numerous and distinctive, and will therefore be first discussed. They are as follows:—

Systolic depression of one or more intercostal spaces to the left of the sternum, more commonly of the third, fourth, and fifth. Skoda believed that systolic depression of intercostal spaces could only occur in cases of adherent pericardium, and that therefore, when present, it was pathognomonic. Friedreich and Traube, however, observed cases where such systolic pittings were present, though the autopsy revealed that there were no pericardial adhesions; they concluded, therefore, that these pittings were due to atmospheric pressure. Normally, systolic recession of intercostal spaces does not occur as a result of the diminution in size of the heart during systole, for the heart presses evenly against the chest walls where it comes in contact with them, and elsewhere the space left by its contraction and consequent diminution in size is filled by the expansion of the overlapping lung.

When the heart is much enlarged, so that the lung tissue which normally overlaps a portion of its surface is pushed aside or is collapsed, systolic depressions of intercostal spaces may occur as the result of atmospheric pressure; for a negative pressure is produced in the thoracic cavity by the diminution in size of the heart during systole, and as the lung is not present to expand and occupy the space left by the receding walls of the heart, the intercostal spaces are forced down by the greater pressure of the atmosphere without. The greater the size of the heart, and the weaker the intercostal muscles, the more marked is this phenomenon. It may most frequently be observed in cases of aortic regurgitation where the heart is considerably dilated and hypertrophied, and is best seen in emaciated persons or young children with cardiac hypertrophy.

But, apart from atmospheric pressure, systolic recession of intercostal spaces may occur as a direct consequence of adherent pericardium. When the pericardium is adherent to the chest wall in front and to the vertebræ and aorta behind, systolic recession of spaces may occur, as the heart, fixed to the unyielding tissues posteriorly, drags in part of the less resistant anterior thoracic wall at each contraction.

When no adhesions exist between the pericardium and thoracic aorta, but only between it and the anterior thoracic walls, systolic retraction of intercostal spaces and of the lower end of the sternum may still occur, as the heart, fixed by the adherent pericardium to the firm unyielding central tendon of the diaphragm, drags inwards the portions of the chest walls to which

it is attached anteriorly; or it may be that the intrinsic force of the contraction of the heart muscle alone, is sufficient to effect this.

Retraction of the Posterior or Lateral Portions of the Thoracic Walls. — In cases of adherent pericardium, marked systolic retraction of some of the lower ribs on the lateral or posterior aspect of the thorax may sometimes be seen. This phenomenon is best seen when the patient is sitting up in a good light, and the movements of the chest are carefully observed from a short distance off, first from the front, and then from the lateral aspect. When a pulsatile movement is seen over the lowest part of the left side of the chest posteriorly, it may at first sight appear to be expansile. On a more careful scrutiny it will be found that there is a tug on the false ribs during the cardiac systole and a sharp rebound during diastole which can be felt as well as seen when the hand is laid flat upon the chest wall at the spot: it is more marked when a deep inspiration is made; it may be seen occasionally not only on the left side but also on the right, especially if the patient leans over to the left.

Here, it is not possible that the heart can be directly fixed to the chest wall at the points of retraction by pericardial adhesions, as the lung tissue intervenes; but the explanation seems to be the following: The heart is, by means of the pericardium, adherent not only to the central tendon of the diaphragm but probably also to a large area of the fleshy or muscular portion of the diaphragm, and, it may be, to the anterior thoracic wall as well; as it contracts it drags

upwards and inwards the less resistant fleshy part of the diaphragm towards the central tendon or anterior chest wall; hence the points of attachment of the digitations of the diaphragm to the lower ribs and costal cartilages are dragged inwards and downwards. It will always be found in such cases that the retracted positions of the chest wall correspond to the floating ribs or costal cartilages of the lower ribs at the points of attachment of the diaphragm. (Systolic recession of the left subcostal angle and epigastrium does not necessarily imply the presence of pericardial adhesions.)

The above is a most important diagnostic sign of adherent pericardium when present, and is quite distinct from recession of the lower ribs in inspiration.

Impeded Descent of the Diaphragm in Inspiration.—Pericardial adhesions may interfere with the descent of the left half of the diaphragm in inspiration; this will be shown by the impaired movement of the upper part of the abdominal wall in the left subcostal angle. If the pericardium is adherent to the chest wall as well as to the heart, the explanation of this impeded descent seems obvious, as the pericardium is normally attached to the central tendon of the diaphragm and to the muscular substance on either side of it for a short distance; where there are no adhesions between the pericardium and chest wall, it is probable that there are abnormal adhesions between the pericardium and the muscular substance of the diaphragm which hinder its descent at their points of attachment.

Vieussens and Morgagni both laid stress on this impeded descent of the diaphragm as a physical sign of adherent pericardium, and Corvisart further called attention to the disturbing effect such adhesions must have on the movement of the heart.

Systolic recession of the site of the apex beat is an important sign when present.

When a definite apex beat can be felt, and systolic recession is seen over its site, this can hardly be due to atmospheric pressure, but implies that the apex of the heart is adherent through the pericardium to the chest wall, and tugs on it during the cardiac systole. For this to occur it is not essential that the pericardium should be universally adherent; a single band stretching between the apex of the heart to the corresponding point of the pericardial sac may give rise to it, if there are also adhesions between the pericardium and chest wall at that point. When there is no palpable apex beat and systolic recession is seen, it may be that the heart is prevented from performing its normal rotatory and forward movement during systole, by adhesions to the diaphragm or vertebral column, or that the cardiac impulse is too feeble to be felt through the adhesions.

Though systolic recession must be rare in the absence of pericardial adhesions, Friedreich has observed one case in which there was marked systolic pitting at the site of the apex, but no pericardial adhesions were found: the case was one of aortic stenosis, and he explains the pitting as due to the fact that the heart, in consequence of insufficient filling of the aorta, could not perform the normal movements,

so that the apex did not come into contact with the chest wall, hence atmospheric pressure caused the depression over its site.

A diastolic shock, when present, is important. It may be felt on palpation with the flat of the hand over the cardiac area generally or at the apex beat or over intercostal spaces in cases in which there is systolic pitting of those areas, or over a larger surface where there is forcible systolic retraction of a portion of the chest wall. It is due to the elastic recoil of the intercostal space or chest wall at the commencement of diastole, as soon as the pulling force exerted during the cardiac systole ceases. For this to occur, the heart must be hypertrophied and acting vigorously.

The Apex Beat may remain fixed, and not alter its position in change of posture of the body, or during deep inspiration or expiration. This is also of great diagnostic importance, as it can scarcely occur unless the apex of the heart is fixed to the chest wall, or the heart is fixed by adhesions, so that it cannot rotate; and nothing, that I am aware of, other than a pericardium adherent both to the heart and chest wall could effect this.

Position of Apex Beat.—The apex beat may be fixed by adhesions unusually high up, perhaps in the fourth space or in the fifth space, in cases in which there is evidence of marked cardiac hypertrophy, so that one would expect it to be situated much lower down. It will usually be displaced outwards in some degree owing to the enlargement of the right ventricle.

It may be absent or extremely feeble, owing to the fact that it does not come into contact with the chest

wall, either because it is prevented by adhesions, or owing to the thickness of interposed tissue; or the heart may be small, and acting feebly.

Heart Sounds.—There is nothing specially characteristic about these. A reduplication of the second sound at the base is common in cases of adherent pericardium; a weak pulmonary second sound, where there is evidence of hypertrophy of the right ventricle is very important, as it indicates that the cause of the hypertrophy of the right ventricle was probably not back pressure through the lungs due to left ventricle trouble, but some intrinsic cause, perhaps adherent pericardium.

The first sound at the apex may be dull or muffled; not infrequently it is prolonged and reduplicated, or a kind of rumbling presystolic murmur accompanies it, which does not, however, indicate the presence of mitral stenosis; this type of presystolic murmur is specially common in children. Of course there may be other murmurs due to mitral incompetence, etc., but these are not specially connected with adherent pericardium.

The area of absolute cardiac dulness will remain unchanged in inspiration and expiration where there are extensive adhesions between the pericardium and chest wall. This phenomenon will be more marked where the pericardium is also adherent to the borders of the lungs.

Increase in the Area of Cardiac Dulness.—The area of cardiac dulness will be considerably larger than normal where there is extensive adhesion between the pericardium and anterior chest wall,

especially in cases where the overlying layer of lung becomes involved in the adhesions, and subsequently becomes collapsed. This will also be the case, to a less extent, where the lung that normally overlaps the heart has been pushed aside without becoming adherent.

Where, during an attack of pericarditis, the area of cardiac dulness has been noted to increase considerably in extent, and after the subsidence of the attack remains permanently increased, it is extremely probable that pericardial adhesions have taken place, fixing the heart in a condition of dilatation. Often, in such cases, a harsh pericardial rub may be heard over portions of the dull area, continuing, perhaps, for some weeks, till the adhesions, previously incomplete, become universal. This does not necessarily imply that adhesions are also taking place between the pericardium and chest wall; but where, at the margins of the cardiac dulness, a pleural friction or pleuro-pericardial friction is also heard, it is probable that such is the case.

CHAPTER III.

PHYSICAL SIGNS (*continued*)—CLINICAL EVIDENCE OF CARDIAC ENLARGEMENT IN CONNECTION WITH ADHERENT PERICARDIUM—ITS CAUSATION.

Enlargement of the Heart.

It is common in cases of adherent pericardium to find the heart considerably enlarged, in the absence of valvular disease, or other cause to account for it. The right ventricle more especially affords evidence of hypertrophy and dilatation, and the apex beat is usually displaced outwards; the left ventricle may be hypertrophied, as shown by the presence of a forcible thrusting apex beat; but not infrequently in such cases the apex beat will be found in the fifth, or even the fourth left intercostal space, instead of being displaced downwards as one would expect, owing to the fact that the pericardial adhesions have prevented the downward displacement. The mechanism by which such cardiac enlargement probably occurs in association with adherent pericardium has already been stated; but it may again be briefly mentioned here, as it is important in connection with the point

under discussion. It is the following: 'The heart becomes dilated during an attack of pericarditis, and before it can again contract down to its normal size the pericardium becomes adherent, and fixes it in this condition of dilatation. The right ventricle, as a rule, suffers more than the left, owing to its thinner walls, and for other reasons stated later on. Cardiac enlargement, therefore more especially hypertrophy and dilatation of the right ventricle, may be a physical sign of great diagnostic significance in cases of adherent pericardium under the following circumstances, to wit: When there is no evidence of valvular disease of the left ventricle, or of kidney disease or other obvious cause which might have given rise to dilatation and hypertrophy of the left ventricle primarily, and secondarily to enlargement of the right ventricle for purposes of compensation; also when there is considerable enlargement of the right ventricle, as evidenced by the increased area of cardiac dulness and displacement of the apex beat outwards, in the absence of valvular disease of the left ventricle or lung disease, such as chronic bronchitis, etc., to account for it.'

Before this statement can be accepted, it will be necessary to prove:—

(1) The occurrence of marked cardiac dilatation in pericarditis.

(2) Persistence of this state of cardiac dilatation after pericarditis, unexplained by valvular disease, and associated with adherent pericardium.

The evidence of several cases, the full notes of which are subjoined at the end of this treatise, will be adduced to prove these two essential points.

I. Cases which show that considerable dilatation of the heart occurs during pericarditis.

Before discussing these cases, a few words must be said on the subject of pericardial effusion, in relation to the increase of the area of cardiac dulness. It is stated by most authors that when the area of cardiac dulness in a case of pericarditis increases to a marked extent and with great rapidity, that this indicates the presence of fluid in the pericardium: a confirmatory sign is said to be that the apex beat becomes less distinct and more diffuse. It is, however, the exception rather than the rule to find effusion of any extent in cases of pericarditis of rheumatic origin, and the increase in the area of cardiac dulness is due to dilatation of the heart, more especially of the right ventricle; the diminution in the force of the apex beat and its more diffuse character is a result of the cardiac dilatation. Dr. Lees has called attention to this point in his paper on the 'Treatment of Pericarditis.' In two cases that I had the opportunity of observing at St. Mary's Hospital, this fact was demonstrated during life, and in one was further verified at the autopsy.

In two cases (A and B of the cases appended), both cases of acute pericarditis, the area of cardiac dulness was observed to increase so rapidly and considerably, and the symptoms became so severe that it was thought that there was extensive effusion in the pericardium, and the operation of aspirating the pericardium was performed. In neither case was any fluid found in the pericardium, though the needle was pushed in gradually and aspiration frequently made: that it was pushed in far enough was shown by the eventual

withdrawal of blood from one of the cavities of the heart, presumably the right ventricle. In the first case (A), paracentesis of the pericardium was performed on May 29, 1893. The patient subsequently died, on August 19th, of the same year. At the autopsy the heart was found to be very much enlarged, chiefly dilated but also moderately hypertrophied, and the pericardium was universally adherent, the pericardial cavity being obliterated. In the second case (B), the area of cardiac dulness—that is, the cardiac dilatation—gradually diminished, and the patient, who was in this instance an adult, made a good recovery, so it is probable that no pericardial adhesions of a serious nature were formed. In a third case (C), the patient, a child of seven, died seven days after the first definite evidence of pericarditis. While the case was under observation the area of cardiac dulness increased to a marked extent: this increase was shown at the autopsy to have been due to dilatation of the heart and not to fluid effusion, as the heart was found to be much dilated and enlarged, and there was no excess of fluid in the pericardium.

II. Cases in which the area of cardiac dulness had increased markedly during an attack of pericarditis, and had not subsequently diminished to any appreciable extent, and in which the autopsy some months later showed the existence of an universally adherent pericardium and a dilated and hypertrophied heart. Cases A, E, and D.

Of this, case A, to which reference has just been made, was an example. The child died rather more than three months after the operation of paracentesis

of the pericardium: the area of cardiac dulness, which was very considerable at the time of the operation, did not diminish appreciably before death; and the dilated and hypertrophied heart with the adherent pericardium were found at the autopsy to be entirely responsible for this abnormal area of cardiac dulness.

The second case (E) was very similar. The patient, a boy nine years old, was admitted to the Children's Hospital, Great Ormond Street, in December, 1893, suffering from pericarditis. From the initial attack he recovered so far that he was able to go to a convalescent home: he then had a fresh attack of rheumatism with carditis, the heart became very much dilated as evidenced by the increase of the area of cardiac dulness, and symptoms of right ventricle failure supervened. The area of cardiac dulness remained permanently enlarged till death, in April, 1894. At the autopsy the pericardium was found to be universally adherent and the heart much dilated. In this case the myocarditis was more severe and extensive in the second attack, hence the cardiac dilatation was more extensive and more rapid, and the break-down of the heart was complete.

In the third case (D) no pericardial rub was audible till four days before death, but the temperature had been above the normal for two months previously, and the action of the heart irregular, so that during this time there was some inflammatory process taking place, probably myocarditis, with pericarditis posteriorly. The heart was somewhat dilated during this period, but became markedly so a fortnight before

death, as evidenced by the increase in the area of cardiac dulness.

At the autopsy the heart was found to be much dilated, and the pericardium universally adherent by recent adhesions which could be easily broken down by the finger, except one of older standing, tough and fibrous, near the apex. In this case both layers of pericardium were coated with a yellow sticky layer of lymph, and were just becoming adherent. This case illustrates what frequently occurs in rheumatic pericarditis: a limited quantity of sticky lymph is exuded, which does not accumulate as fluid in the pericardium, but forms a sticky gelatinous coating to both layers of the pericardium, favouring the formation of adhesions.

From a consideration of these cases, therefore, one is justified in concluding that cardiac enlargement, unexplained by valvular disease or other causes, may be associated with and indirectly due to adherent pericardium; hence it may under these circumstances be an important physical sign of that condition, or, failing that, give rise to a suspicion of the presence of adherent pericardium which other physical signs and symptoms may confirm.

As further evidence, the notes of three cases are appended, in which, from the size of tho heart, supported by other physical signs, a diagnosis of adherent pericardium was made, though no history of pericarditis was obtainable, and was proved to be correct by the autopsy. In these cascs there was, however, valvular disease of tho heart as well. See notes of cases K, and L, and J. The commentaries attached

to the notes of the cases referred to will bring out the chief points illustrated by them.

Sudden Diastolic Collapse of Cervical Veins.—This phenomenon, mentioned by Skoda and Ajka, has been specially studied by Friedreich, who regards it as of great diagnostic value when accompanied by systolic retraction of spaces. He states that it is quite different to venous pulsation, seen in cases of tricuspid incompetence. He explains it as due, partly to a sudden increase in the size of the thoracic cavity immediately following the relaxation of the heart in diastole, whereby it allows the diaphragm to descend and the thoracic walls to expand, at the points where the pericardial adhesions had during the cardiac systole pulled on them and impeded their movements; partly, also, to the fact that the heart, being fixed by adhesions to the diaphragm, descends with it, and causes elongation of the large venous trunks. Hence, as a result of both processes, an aspiratory action takes place, causing such acceleration of the flow of blood from the veins of the neck, and consequent collapse of their walls during the cardiac diastole.

It is difficult to believe that the force of the cardiac systole, unless the heart was much hypertrophied and the subject had remarkably elastic chest walls, could possibly exert sufficient traction to cause a definite aspiratory effect in that way. Again, if the diastole occurred during the phase of expiration when the diaphragm was rising and the chest walls collapsing, the explanation given by Friedreich would lose its point.

Systolic emptying of the veins of the neck

was observed by François Franck, in a case of adherent pericardium, which he ascribed to an aspiratory periventricular effect caused by the adhesions.*

Systolic emptying of an enlarged vein on the front of the chest was observed in the case of Eliza F. (case K), described later. An enlarged vein to the right of the sternum was observed to empty during systole, and fill during diastole of the heart. The explanation suggested was, that the pericardium adherent to the heart and chest wall dragged apart the walls of the internal mammary vein during systole, causing a suction action, so that blood was drawn into its lumen from the afferent veins during systole.

In this case the autopsy showed the pericardium to be universally adherent to the heart, which was greatly hypertrophied, and also to the chest walls over a large area.

Pulsus Paradoxus, or pulsus inspiratione intermittens, has been observed to be present by Kussmaul and others in some cases of adherent pericardium. He believed its presence to be of great diagnostic importance, as indicative of indurated mediastino-pericarditis. He considered the occurrence of the pulsus paradoxus in such cases as due to the fact that indurated fibrous cords, the result of the mediastino-pericarditis encircled the aorta, and dragging on it constricted its lumen during inspiration. Three cases of his, illustrating this point, are described in Ziemssen's 'Encyclopædia of Medicine,' vol. vi. p. 649, sqq. But the pulsus paradoxus can be produced

* 'Traité de Med.,' Charcot, Bouchard, Brissaud, 1893, tom. v. p. 56.

in healthy persons by holding the breath in inspiration, or by rapidly taking several deep breaths, as Sommerbrodt has observed. Hence we cannot attach any diagnostic significance to it.

Irregularity of Pulse.—This is mentioned by Boerhave, Morgagni, and other ancient writers as occurring in cases of adherent pericardium. It is by no means of necessary or common occurrence till signs of cardiac failure supervene. As irregularity of pulse may occur in cases of mitral incompetence and of cardiac failure arising from any cause, it is not of great importance in connection with adherent pericardium.

CHAPTER IV.

SYMPTOMS—RELATION OF SYMPTOMS TO PHYSICAL SIGNS — INFERENCES TO BE DRAWN FROM EXCESSIVE SEVERITY OF SYMPTOMS—EFFECTS OF ADHERENT PERICARDIUM ON THE RIGHT VENTRICLE—REASONS FOR SERIOUS EMBARRASSMENT OF RIGHT VENTRICLE BY ADHESIONS.

Symptoms in Cases of Adherent Pericardium.

The symptoms in themselves are not in any sense characteristic. They are usually some of those arising from cardiac failure, and especially from the giving way of the right ventricle ; namely—

Shortness of breath on slight exertion.

Pain and tenderness over region of liver from hepatic engorgement.

Vomiting from congestion of gastric mucous membrane, secondary to congestion of liver.

Œdema of lower extremities.

Ascites.

Other symptoms may be present, such as—

Pain over præcordial area.

Palpitation.

Faintness, and pallor of face.

Relation of Symptoms to Physical Signs.

The physical signs or the symptoms of adherent pericardium, few of which may be present, are often in themselves insufficient to allow of a diagnosis of this condition being made, or even to arouse a suspicion of its presence; but in a case of morbus cordis valuable help may be derived from careful consideration of the physical signs and symptoms together, and by balancing the former against the latter, so that the question is raised, 'Do the physical signs present afford evidence of sufficient disease to account for the symptoms that arise?' When the symptoms are those of right ventricle failure, and are more severe than the physical signs present would lead one to expect, adherent pericardium must be thought of. It is the right ventricle more especially that is seriously embarrassed and hampered by pericardial adhesions, hence their presence may account for the unexpected break-down of the right ventricle in cases where the physical signs seem to indicate that the valvular lesion is slight, or where there is no indication of the presence of valvular disease or other cause to account for the symptoms of right ventricle failure.

In support of this statement, and to illustrate its meaning, the history of several cases will be given.

Clinical Evidence of the Serious Effects of Adherent Pericardium on the Right Ventricle —Inferences to be drawn from Excessive Severity of Symptoms.

Two cases, A and E, which will first be considered, may be taken together, as they are very similar in many points. They both illustrate the serious effects of adherent pericardium on the right ventricle, causing its break-down, and, ultimately, death of the patients from right ventricle failure. In these two cases, in which the patients were both children under twelve years of age, pericarditis was seen to result in universal adhesion of the pericardium while the children were under observation in hospital; this result was also verified at the autopsy. In both instances the children recovered from the primary attack of pericarditis, though the period of convalescence was protracted. In case A, the pericardial rub was audible for some weeks after it was first heard; in case E, it was only audible for a few days. In both cases, after a period during which the child seemed to improve, symptoms of right heart failure supervened, without any apparent cause, as in the one case (A) the child was in hospital the whole time, while in the other case (E) he was at a convalescent home and well looked after, so that in neither instance was there a possibility of undue exercise or exertion having brought about the relapse. For a certain period the right ventricle had been able to compensate for the slight lesions of the mitral

valve, and during this period the child improved in both cases; then the right ventricle began to fail, and soon the liver became enlarged; œdema set in, and steadily increased till death, which occurred some weeks after.

What, then, occasioned the break-down of the right ventricle? Clearly not backward pressure through the lungs due to the trifling mitral regurgitation, as there was no cyanosis or evidence of pulmonary congestion. It could not have been merely the cardiac dilatation resulting from the pericarditis, or in both cases the child would have died immediately after, or during the attack itself.

Presumably it was due to the adherent pericardium, as follows: The right ventricle at first was able to contract and do its work during the period in which the pericardium was becoming adherent, as the adhesions at first were loose, and could be easily broken down, and so allowed of a certain degree of movement. As the adhesions became firm and organized into fibrous tissue, a point was reached when the right ventricle was unable to do its work efficiently owing to these adhesions; hence arose symptoms of right heart failure, viz. enlarged liver, œdema, etc. These became more and more pronounced as the adhesions became firmer and the right ventricle more dilated and its contractile power feebler, and ultimately the child died from failure of the right ventricle.

In case E, an attack of pleurisy supervened, which threw extra work on the right side of the heart, and so precipitated the break-down of the right ventricle. In these two cases adherent pericardium was the

chief, if not the determining cause of the break-down of the right ventricle.

In a third case, J, it was a contributory and important cause of right ventricle failure in the same way. A child, E. T., aged 9, was admitted to the Children's Hospital in December, 1893. On admission, she complained of shortness of breath and pain in the præcordial area; the pulse was 130, temp. 100, resp. 32. The heart was dilated and somewhat hypertrophied, the apex being in the sixth space, one inch outside the vertical nipple line, and the area of cardiac dulness extending to the right border of the sternum. A double mitral murmur was audible at the apex. Neither on admission nor at any time was a pericardial rub audible; probably when she came into hospital the pericardium was already adherent. Throughout her stay in hospital her temperature was above the normal, varying from 102·4 to 99·4, and at different times she had eruptions of erythema marginatum and of rheumatic nodules, evidencing the presence of active rheumatism. On January 25th the presence of active endocarditis was also proved by the appearance of a diastolic murmur heard at the aortic cartilage and down the edge of the sternum; during the next few days she got rapidly worse, the liver increased in size, the feet became œdematous, and there was pulsation of the veins in the neck. She died on February 1. The pericardium was found at the autopsy to be universally adherent, the adhesions being firm but not yet transformed into tough unyielding fibrous tissue; the valvular lesions, both

aortic and mitral, were slight, and could not in themselves have given rise to right ventricle failure from back pressure. As there was active endocarditis, and, doubtless, accompanying myocarditis, it cannot be claimed that adherent pericardium was in this case entirely responsible for the break-down of the right ventricle; but that it was a contributory, if not the principal cause, is, I think, shown by the history of the case. On admission there was evident embarrassment of the right ventricle, as shown by the epigastric pulsation, the enlarged area of cardiac dulness, and enlarged liver. The slight mitral lesion could not have given rise to this by back pressure; the adherent pericardium was mainly responsible for this embarrassment of the right ventricle, which, however, was able to do its work in an imperfect fashion till the attack of acute endocarditis supervened; then either the extra work thrown on it by the aortic incompetence which ensued as a result of the endocarditis, or more probably because it was further weakened by damage of its muscular fibres by myocarditis accompanying the endocarditis, it broke down, and hence resulted further enlargement of liver, pulsation of jugular veins, œdema of feet, and other signs of right ventricle failure.

In another case, H, a woman, was admitted to St. Mary's Hospital with œdema of legs, enlarged liver, and pulsating jugular veins—symptoms of right ventricle failure. On examination, the apex beat could not be seen or felt, but a weak first and second sound could be heard in the fifth space in nipple line; no murmurs could be heard at base or apex on

auscultation, but over the tricuspid area a systolic murmur was heard; there was also marked epigastric pulsation. There was thus no evidence of hypertrophy or dilatation of the left ventricle, or of valvular disease, but it was clear that the right ventricle had broken down, and was seriously embarrassed. This could not be due to back pressure from left ventricle failure, nor was there any pulmonary disease to account for it; adherent pericardium was thought to be the cause, and this diagnosis was further rendered probable by the fact that there were marks of old leech bites over the præcordial area. She stated that the leeches had been applied when she had rheumatic fever, so presumably she had suffered from pericarditis. With rest and treatment she improved, and was able to leave the hospital. Unfortunately she has been lost sight of, but the history of the case, as far as is possible without an autopsy, is strong evidence in favour of adherent pericardium as the cause of the right ventricle failure.

These four cases, more especially the first two quoted, demonstrate the serious effects of adherent pericardium on the right ventricle, causing its breakdown, and, in three instances, death of the patient with all the symptoms of right ventricle failure. From this, one may deduce the following corollary: 'That when symptoms of right ventricle failure supervene in cases in which there is no evidence of left ventricle failure due to valvular disease or kidney mischief, constant high tension, or other obvious causes, or of lung disease, such as chronic bronchitis, etc., to account for their appearance, the presence of

adherent pericardium should be suspected as the cause, and other indications of it carefully sought for. So, too, in valvular disease of the left ventricle, in which the lesion is judged to be slight, and compensation breaks down unaccountably, adherent pericardium should be thought of.'

As an illustration of the way in which a diagnosis of adherent pericardium was arrived at in this way in an adult, where no history of pericarditis could be obtained, the following case may be quoted :—

Case G.—The patient, a man aged about twenty-seven, was admitted to St. Mary's Hospital in May, 1891, with œdema of lower extremities and of lower part of trunk. He had enjoyed good health up to a year previous to admission, when, he said, he had an attack of 'pleurisy' on both sides; since that time he had always suffered from shortness of breath and œdema of legs, and had not been able to get about. On admission there was œdema of both legs and of the side of the trunk on which he lay; the liver was enlarged, the veins in the neck were distended. The apex beat could not be seen or felt, and no murmurs were audible at base or apex; the urine contained no albumen nor casts. The symptoms were severe, and were those evidencing failure of right ventricle and obstruction to the venous return; the physical signs gave no indication of valvular disease or of lung disease which might have given rise to embarrassment of the right ventricle by backward pressure through the pulmonary system or obstruction to the flow of blood through the lungs. Nor were there any indications of pulmonary congestion. The pulse

was small, and the apex beat impalpable, so that there was evidence of deficient power in the left ventricle, but the cause of this was not at first obvious. Rest and treatment by drugs failed to restore the compensatory balance, or to make any appreciable effect on the œdema.

Adherent pericardium seemed to be the only possible explanation of the obvious embarrassment of the right ventricle as evidenced by the symptoms. The only physical signs which could be said to be in favour of this diagnosis were the absence of apex beat with increased area of cardiac dulness. The diagnosis of adherent pericardium was made in life mainly by a consideration of the symptoms and physical signs together, and by balancing the former against the latter. Adherent pericardium was thought to be responsible for the serious embarrassment of the right ventricle which could not be explained by the physical signs present. This diagnosis was found to be correct at the autopsy some months later, when the pericardium was found to be universally adherent and the right auricle was seen to be so strangled by the adhesions that its cavity was almost obliterated. There was no evidence of any damage to valves by endocarditis.

Reasons why Pericardial Adhesions affect the Right Ventricle more seriously than the Left.

There are many reasons why one would expect the right ventricle to suffer more severely than the left when hampered by pericardial adhesions.

Firstly, the walls of the right ventricle are thinner,

and its muscular contractile power is therefore less than that of the left ventricle; hence it would be less able to free itself from adhesions while in process of formation, and when adhesions were formed would be less competent to contract against them and fully complete the systole, than the stronger left ventricle.

Secondly, when we consider the effects of myocarditis on the two ventricles, it is clear that the thin walled right ventricle would have less chance of recovery than the left ventricle, where there is a much greater thickness of muscular substance. Further, when the myocarditis is limited to the superficial layers of muscle, being simply a spread of the inflammatory process from the inflamed pericardium to the subjacent muscle, the extent of muscle damaged would be a much larger proportion of the whole in the thin-walled right ventricle, than in the thicker left ventricle.

Thirdly, when we consider the normal movements of the heart, and see how they would be affected by adherence of the pericardium, it will be seen that the movements of the right ventricle will be more seriously interfered with than those of the left.

The normal position of the heart in man, as seen post-mortem, is thus described by Sibson: * 'The right ventricle, when exposed to view in the front of the heart, presents a pyramidal shape. The base of the pyramid is formed by the lower boundary of the ventricle which rests on the central tendon of the diaphragm, and extends with slight obliquity downwards and from right to left, from the right auricle to the apex of the left ventricle.'

* 'Sibson's Works,' edited by Ord, vol. iii. p. 130.

On p. 113 of the same volume the systole of the ventricles, as observed in the living dog and ass, is described by Sibson as follows: 'During systole the ventricles, when looked at in front, contract from all sides towards a given centre which is situated on the right ventricle, a little to the right of the septum, about midway between the origin of the pulmonary artery and the lower boundary of the ventricle where it rests on the diaphragm. The contraction of the right ventricle, owing to its position in the front of the heart and consequent exposure, is seen to be marked and vigorous. The whole *right margin of the ventricle* at its juncture to the auricle *moves extensively from right to left*, while its left margin, at the longitudinal furrow, or septum, between the ventricles, moves to a comparatively slight degree from left to right. At the same time, the top of the ventricle, at the origin of the pulmonary artery, descends, while its *lower border, where it rests on the diaphragm, ascends.* The point of rest towards which these various movements converge corresponds closely with the attachment of the anterior papillary muscle.'

A little further on he continues: 'The vigorous contraction of the left ventricle is only visible at its apex and along its left border, since the rest of the cavity is hidden by the right ventricle. The apex has a revolving movement, upwards, forwards, and to the right. The left border of the ventricle, like the apex, moves forward and to the right; but while the portion of the ventricle near the apex ascends, the portion near the base descends. . . . When we remove the ribs and look at the heart from the left so as to

obtain a profile view, we see that the whole left ventricle moves forward during systole, the posterior wall advancing more than the anterior; and that the base of the ventricle descends while the apex ascends, so that apex and base approximate.'

When, in connection with these movements, we also consider the firm attachment of the pericardium to the central tendon of the diaphragm, on which the right ventricle rests, it is obvious that, when the pericardium is adherent universally to the heart, it also binds down the right ventricle to the diaphragm; where the pericardium is also adherent to the diaphragm over an abnormally large area as a result of pericarditis, this fixation is more extensive and is more serious in its results: under these conditions the contractile movements of the right ventricle must be seriously hampered, especially the movements from below upwards and from right to left. Again, when adhesions exist between the pericardium and chest wall, it is usually the walls of the right ventricle which become fixed to the sternum and costal cartilages. More rarely do the walls of the left ventricle become adherent to the vertebral column posteriorly: the normal attachment of the pericardium by fibrous bands to the manubrium and ensiform process of the sternum favours the production of adhesions anteriorly, and, as the right ventricle lies in front, a large proportion of its substance may become adherent to the chest walls, which could not be the case with the left ventricle. The apex of the left ventricle may become fixed by adhesions to the chest wall; this would hamper the contractile movement of the approximation

of the apex to the base during the systole of the heart, and would interfere with the rotatory and other movements of the heart as a whole, but would not be so serious as fixation of a large portion of its walls.

Some Points of Difference in the Symptoms of Right Ventricle Failure when due to Pericardial Adhesions, and when Secondary to Valvular Disease of the Left Ventricle.

In cases of right ventricle failure attributable to adherent pericardium there is no cyanosis, though the respirations may be hurried, and there may be some dyspnœa; there may be an entire absence of dyspnœa though the other symptoms are severe; there is usually no congestion or œdema of the lungs.

The dyspnœa, when present, is probably due to deficient supply of blood to the lungs and a feeble pulmonary circulation, owing to the failing powers of the right ventricle; and in such cases the pulmonary second sound will be feeble instead of accentuated. As the right ventricle failure is due to the embarrassing effect of pericardial adhesions, and not to back pressure through the lungs and obstruction to the pulmonary circulation, one would not expect to find evidence of pulmonary congestion, as in cases where the right ventricle failure is secondary to severe valvular disease of, or the breakdown of the left. Hence, the difference in symptoms may be of great diagnostic value in the absence of serious valvular disease of the left ventricle, or evidence of left ventricle failure.

CHAPTER V.

DIAGNOSIS—PROGNOSIS.

Diagnosis.

When there is a trustworthy history of pericarditis at some period, the question of the possibility of the existence of pericardial adhesions will always arise, and physical signs indicative of its presence should be carefully sought for. In the absence of history of pericarditis, there will be no presumptive evidence in favour of adherent pericardium; but in cases of valvular disease dating from childhood, evidence of pericardial adhesions should be looked for, inasmuch as pericarditis so commonly accompanies endocarditis in children, as Dr. Sturges pointed out. When adherent pericardium is present as a complication of valvular disease, the valvular lesions may be thought sufficient to account for the symptoms that arise, and its presence may not be suspected.

Adherent pericardium may give rise to no physical signs or symptoms during life to indicate its presence. In such cases it will usually be found at the autopsy that there are no adhesions between the pericardium

and chest wall, and that the heart is of normal size. In most cases it is difficult to arrive at a certain diagnosis, especially in the absence of any history of pericarditis at any time; but the following points may be especially considered in regard to diagnosis.

Systolic Recession of Spaces.—Skoda thought that systolic recession of intercostal spaces was diagnostic of pericardial adhesions. Friedreich showed that such recession of spaces might result merely from the effects of atmospheric pressure; he, however, attached great importance to this recession of spaces, where accompanied by diastolic collapse of cervical veins previously discussed. With regard to pulsation or systolic recession of intercostal spaces, when it is present adherent pericardium must be taken into consideration as a possiblo cause of it; but when the heart is hypertrophied, especially in cases of aortic regurgitation, no great diagnostic significance can be attached to its presence, as it is under such circumstances likely to be due simply to the effects of atmospheric pressure.

The Apex Beat.—Systolic recession of the area over the apex beat is of more significance, and, when the apex beat is also immovable in deep inspiration, is probably diagnostic of adherent pericardium. A further proof would be the presence of a 'diastolic shock' felt over the retracted area. The position of the apex beat in the fourth or fifth space, when there is evidence of considerable hypertrophy of the left ventricle, instead of in a lower space, as one would expect, should lead to tho suspicion that it may have been fixed thero by pericardial adhesions. Absence

of, or weakness of, the apex beat is in itself of no diagnostic value; but when occurring in cases where there are symptoms of right ventricle failure for which there is no obvious cause, it may be of considerable importance. Cf. cases G and H.

Systolic retraction of the lower ribs on the lateral or posterior aspect of the thorax is of the greatest importance, and can scarcely be due to anything else than pericardial adhesions.

Enlargement of Heart.—When the heart is found to be dilated and hypertrophied in a case in which, as regards the left ventricle, there is no evidence of valvular disease or kidney disease, high tension, or other obvious cause, or, as regards the right ventricle, of lung disease to account for it, or where, in a case of valvular disease, the hypertrophy of either right or left ventricle is greater than the valvular disease would lead one to expect, adherent pericardium should be thought of as a possible cause of this enlargement. The reasons for this have been fully discussed elsewhere, so they will not be further enumerated here.

Relation of Symptoms to Physical Signs.—In all cases of valvular disease of the heart, an attempt should be made to estimate the extent of the lesion. This can be done by a careful observation and consideration of the physical signs present; the symptoms also should be carefully noted, and the question asked, whether they are such as the nature of the lesion would lead one to expect, or of greater severity; secondly, in cases where compensation has broken down, it should be ascertained whether this was due

to some imprudence or some intercurrent pulmonary trouble, or whether there was apparently nothing to account for it. When symptoms of cardiac failure, more especially of right ventricle failure occur of greater severity than the physical signs present would seem to warrant, or where compensation breaks down unaccountably, adherent pericardium must be suspected. When rest and suitable treatment fail to give relief, provided the patient is not of advanced age or thoroughly broken down, this affords further evidence in favour of adherent pericardium, and other confirmatory physical signs of it should be carefully looked for.

When with symptoms of right ventricle failure there is an absence of cyanosis or evidence of pulmonary congestion and œdema, adherent pericardium must be thought of as a possible cause of the break-down of the right ventricle. It will at once be asked, How is the extent of the valvular lesion to be estimated? This is too large a question to discuss here, and each case must be judged on its merits; but for means by which this estimation may be made, I would refer to 'The Harveian Lectures on Prognosis in Heart Disease,' by W. H. Broadbent, *Brit. Med. Journ.*, 1884, pp. 139, 256, 400 and 449, where the subject is fully discussed, and which I have taken as my guide.

In cases of pericarditis which can be kept under observation throughout the attack, the following are the indications which would lead one to suspect that pericardial adhesions are in process of formation.

1. Prolongation of the attack of pericarditis as evidenced by the presence of a harsh rub for some weeks over part of the præcordial area.

2. Permanent enlargement of the area of cardiac dulness after the subsidence of the attack of pericarditis.

3. Prolonged convalescence, during which time the patient remains thin, pale, apathetic, and incapable of any exertion. During this period of convalescence the temperature may be irregular, and attacks of dyspnœa supervene; a pleuritic rub may be heard, as intercurrent attacks of pleurisy are not unlikely to occur, and give rise to adhesions between the lung and chest wall; for all the serous membranes may be affected by the same chronic inflammatory process which is taking place in the pericardium. Cf. cases A, G, H, for occurrence of pleurisy.

4. The occurrence of symptoms of right ventricle failure, after a period of temporary improvement, there being no apparent cause for the break-down of the right ventricle. Cf. cases A and G.

In the case of children, in whom pericarditis is far more common than in adults, the physical signs of adherent pericardium, when they supervene, are usually more obvious than in older persons, owing to the yielding nature of the thorax, and the absence of much muscular or adipose tissue over the chest wall; hence retraction and pulsation of spaces, recession of epigastrium, etc., are more easily seen.

Prognosis.

The prognosis in cases of adherent pericardium is by no means so absolutely unfavourable as Corvisart, Hope, and others have made out. There are numerous cases on record in which the existence of extensive pericardial adhesions has apparently given rise to no ill effects. For example, on reference to the post-mortem records of St. Mary's Hospital, I found that in one case, where a universally adherent pericardium was found at the autopsy, the man had up to the age of sixty-five enjoyed very good health and been able to do his work regularly; he died at the age of seventy-one with symptoms of right heart failure, so it is possible that the adherent pericardium may have precipitated death, but not till the heart was becoming enfeebled by old age. In another case, the patient died from hemiplegia at the age of fifty-seven; during life there were no symptoms to indicate that he had experienced any ill effects from the existence of the adherent pericardium. In a third case, which I had the opportunity of observing during life, there were no symptoms or physical signs which could lead one to suspect the presence of adherent pericardium. The man, aged thirty-six, was admitted for ascites and jaundice, and, as there was a well-authenticated history of chronic alcoholism and other indications of cirrhosis of the liver, the diagnosis made was alcoholic cirrhosis of liver, which was found to be correct at the autopsy, the substance of the liver being very extensively replaced by fibrous tissue, and the portal vein much

dilated. Besides this, he was found to have a universally adherent pericardium. This had given rise to no symptoms or physical signs during life which could be attributed to the presence of pericardial adhesions, and did not appear in any way to have been instrumental in causing his death.

In these three cases, however, there were no abnormal adhesions between the pericardium and chest wall or diaphragm.

But in far the greater proportion of cases adherent pericardium is a serious lesion, especially when it is also adherent to the chest wall. Where it exists as a complication of valvular disease it is still more likely to prove fatal eventually, by so hampering the right ventricle as to prevent its recovery when once compensation has broken down; possibly in less severe cases recovery may take place once or twice after symptoms of right ventricle failure have set in, but each time the heart is left a little more damaged than before, and eventually rest and treatment fail to make any impression on the symptoms, which progressively increase till death ensues.

It would appear that it is only in cases in which the heart is of normal size, and the pericardium is not adherent to the chest wall, or to a large area of the diaphragm, that adherent pericardium gives rise to no serious consequences. In other words, one is forced to the somewhat unsatisfactory conclusion that it is only in cases in which adherent pericardium gives rise to no definite signs or symptoms in life to indicate its presence that the prognosis is good; such is the evidence which the unexpected discovery of

adherent pericardium post-mortem puts forward in many cases. When there are physical signs and symptoms during life to indicate its presence, the degree of unfavourableness of the prognosis depends mainly on whether there is valvular disease as well, and if so, whether compensation has broken down for no apparent reason, or owing to undue exertion or intercurrent pulmonary trouble. In the former case treatment will probably fail to do any good, in the latter it may perhaps restore for a time the compensatory balance.

In conclusion, my thanks are due to the late Dr. Sturges, Dr. Barlow, and Dr. Lees, and Sir William Broadbent for kindly allowing me to quote and make use of the cases under their care which I had the opportunity of observing while acting as House Physician at the Children's Hospital and St. Mary's respectively. Appended are the notes of cases referred to in this work.

CHAPTER VI.

BRIEF SUMMARY OF CASES—NOTES OF CASES IN DETAIL, WITH COMMENTARIES.

It may be useful here to give a brief summary of the chief points of interest in the cases appended below. Taken collectively, they form a kind of life history of adherent pericardium; thus cases C and D illustrate its earliest origin in pericarditis; cases A and E the process of formation of adhesions followed by their gradual organization into fibrous tissue; cases G, K, L, M are instances of mature firm adhesions already organized into unyielding tough fibrous tissue. Taken individually, they serve to illustrate and prove several of the most important conclusions in this treatise. Thus, cases A, B, C, D demonstrate the cardiac dilatation that occurs in cases of pericarditis as a result of the accompanying myocarditis. Cases A and E show how pericardial adhesions, becoming organized into fibrous tissue, prevent the heart from again contracting down to its normal size. Cases A, E, G and M demonstrate the serious effects of pericardial adhesions on the right ventricle more especially. Cases G, H, K, L and M prove that a

correct diagnosis of adherent pericardium may be made during life in the absence of any history of pericarditis, partly by physical signs, but mainly by a careful consideration of the symptoms and physical signs together, and by the fact that the symptoms were more severe and persistent than would be accounted for by the physical signs present. The commentaries attached to the cases further bring out the chief features of interest in each case.

Case A.

Case of Pericarditis in which paracentesis of the pericardium was performed and which ended in universal adherence of the pericardium.

ANNIE M., æt 8.

Admitted to St. Mary's Hospital April 13, 1893. Died August 19, 1893.

History.—She had an attack of rheumatism in January, 1893, and for some time after she had pain in her joints and chest; she has never been well since. Father and mother have both suffered from rheumatism.

Condition on Admission.—Pale, anæmic, fairly well nourished girl. Pulse, 76. Respiration, 20. Temperature, 99° F.

Cardio-vascular System.—Pulse 76, regular in force and frequency: wave ill-sustained; artery of medium size, easily compressible, felt between the beats.

Heart.—Apex in sixth space ½ inch outside nipple

line; the beat is diffuse but forcible: pulsation is seen in third, fourth and fifth spaces to left of sternum.

The *cardiac dulness* extends inwards to right margin of sternum; upwards to fourth rib and outwards ½ inch outside nipple line.

Systolic and presystolic thrill felt over area of apex beat.

On *auscultation* a systolic murmur is heard at the apex conducted into the axilla, also a rumbling presystolic murmur. There is accentuation of the pulmonary second sound at the base, and reduplication of second second to left of sternum. The first sound is low-pitched and rather prolonged; the second sound at the apex is well heard.

Liver edge is felt one inch below costal margin.

Lungs nothing abnormal.

Urine contains no albumen or sugar.

The child improved somewhat and went on well till—

May 10*th*, when the temperature rose to 103, pulse, 160, respiration, 52, and she vomited three times during the morning; on auscultation a pericardial rub was heard at the apex.

May 11*th*.—Vomiting still persistent; pericardial rub more extensive, heard to left of sternum about level of fifth and fourth ribs.

May 13*th*.—Vomiting still troublesome; nutrient enemata ordered, and no food given by mouth; pain in præcordium severe; three leeches applied, which gave relief. Loud scratching rub audible to left of sternum as far upwards as third rib.

May 15*th*.—Vomiting has ceased; she seems a little

better. Liver is enlarged, and the edge is felt two inches below costal margin. The area of cardiac dulness is also increased; it extends upwards to third rib; inwards to just beyond right margin of sternum; outwards 2 fingers' breadth beyond nipple line. A triple cantering rhythm is audible over the lower end of sternum like the puffing of a locomotive, and a friction rub is heard over nearly the whole of the dull area.

May 25th.—Has been getting gradually worse; considerable dyspnœa; much distressed; leans on left side of cot. The cardiac dulness is increased, and extends inwards to 1 f. b. beyond the right border of the sternum and upwards to second left space; outwards 1½ f. b. beyond nipple line. A pericardial friction rub is heard over the whole of the dull area.

The liver edge can be felt one inch above umbilicus.

May 28th.—Much worse; sits up and leans forward with head resting on left arm, which is supported by the bed-board; she cannot rest in any other position; there are signs of fluid at left base; on aspiration, 1½ ounces of fluid were removed from the left pleural cavity.

May 29th.—Rather worse; area of cardiac dulness increasing; the dulness extends upwards to second rib; inwards to one inch beyond right margin of sternum nearly as far as right nipple; outwards it merges into dulness at base of left lung. Friction rub still heard at apex, but not at base.

The position assumed by the child, her dyspnœa and anxious expression, together with the increased area of cardiac dulness were thought to indicate that fluid in the pericardial cavity was causing the distress.

An incision was made in fifth left space close to sternum, and the pericardium was exposed, and an aspirating needle passed in: no fluid was withdrawn till it was pushed in far enough to penetrate the heart, when blood was withdrawn. Hence it did not appear to be excess of fluid in the pericardium, but cardiac dilatation, which was giving rise to the symptoms.

June 5th.—Patient is rather better; the area of cardiac dulness is still the same as on May 29th, and is not diminished at all in extent. Friction rub audible only at apex.

June 7th.—There is œdema of the dorsum of both feet; the liver extends downwards to one inch below the level of umbilicus; the area of cardiac dulness is slightly increased.

From this time forward the child got gradually worse, and the œdema of the lower extremities increased; the liver remained of great size, its edge being felt one inch below the umbilicus. Ascites also supervened, and the amount of fluid in the left pleural cavity increased.

The area of cardiac dulness did not diminish, but slightly increased before death.

She died on August 19th, the œdema of lower limbs and trunk being very marked.

Autopsy.

Body of child with extreme œdema of lower extremities and trunk.

Pericardium universally adherent to heart by adhesions which could be broken down with difficulty

by the finger for the most part, but which were tough and fibrous in places. The pericardium was also partly adherent to the chest wall.

Heart greatly enlarged; right auricle much dilated; right ventricle much dilated and hypertrophied; Tricuspid orifice admitted three fingers; valves normal.

Left ventricle dilated and hypertrophied; left auricle dilated.

Mitral orifice admitted three fingers; valves thickened at edges by old vegetations.

Aortic and pulmonary valves normal.

Liver much enlarged; 'nutmeg' on section.

Lungs and pleura.—Some fluid in left pleural cavity; lungs congested and œdematous.

Commentary.

The child came into the hospital for cardiac symptoms, and on admission the heart was found to be enlarged; the presence of a systolic murmur at the apex indicated mitral regurgitation, which would account for some degree of enlargement of the left ventricle as a result of this, and also for some enlargement of the right ventricle as compensatory hypertrophy; but the enlargement seemed too great to be accounted for simply as a compensatory hypertrophy, and was probably due to the heart having been left dilated in a previous attack of carditis, and not having recovered before some adhesions were formed, and it had extra work thrown on it by the mitral lesion. Shortly after admission, the child developed

pericarditis, and during the attack the heart dulness increased so considerably, and the symptoms were so severe, that it was thought effusion into the pericardium might be the cause of this; aspiration of the pericardium proved this was not so. The increased area of cardiac dulness was presumably due therefore to cardiac dilatation, more especially dilatation of right ventricle; later on the increase in size of the liver and subsequent onset of œdema evidencing failure of the right ventricle proved this to be the true explanation.

The area of cardiac dulness which had increased considerably during the pericarditis did not diminish before death, when the autopsy showed that it was due to the enlarged heart to which the pericardium was universally adherent, there being no fluid in the pericardial cavity, which was quite obliterated.

The symptoms of right heart failure, viz. œdema of lower extremities, enlargement of liver, etc., gradually became more and more pronounced, and ultimately she died from the effects of the break down of the right ventricle, the œdema of the lower extremities and of the whole body being extreme, and the size of the liver enormous. Thus the attack of pericarditis was observed while the child was in hospital to result in adherent pericardium, which ultimately proved fatal; the heart during the attack of pericarditis became dilated; before it recovered the pericardium became adherent; the heart was not able to undergo sufficient hypertrophy to overcome the hampering effects of the adherent pericardium; the right ventricle was especially embarrassed and broke down; hence resulted enlarged liver, tricuspid regurgitation, and

extensive œdema of nearly whole body, and finally death.

In this instance, on the admission of the child to the hospital, the heart was thought to be more seriously crippled than the mitral lesion would account for, and a previous attack of pericarditis was thought to be responsible for this. The finding, at the autopsy, of tough fibrous adhesions which could not be stripped from the heart without tearing its substance, side by side with others of later dates, less tenacious and more elastic, confirmed this hypothesis.

Case B.

Case of Pericarditis in an adult in which aspiration of the pericardium was performed. Recovery.

ARTHUR, F., æt. 30.

Admitted to St. Mary's Hospital, January 13, 1894. Discharged, March 5, 1894. Admitted complaining of pain in chest and back, and of cough with expectoration.

History of Illness.—Was taken ill on January 6th, a week ago, with rigors followed by profuse sweating, and with pains in limbs. Lost his appetite, and three days later coughed a good deal, and had severe pain in chest. He is a coachman, and has never had any serious illness before.

Present Condition.—Is a well-nourished man, lies on his back, has frequent cough, and complains of pain in chest. Pulse, 96. Respiration, 32. Temperature, 99·8.

Cardio-vascular System.—Pulse, 96; regular in force and frequency. Artery large, wave short.

Heart.—Apex beat cannot be seen or felt.

On *percussion* (January 15th) cardiac dulness is found to extend upwards to upper border of 3rd rib; inwards 1 f. b. to right of sternum; outwards 1 f. b. outside vertical nipple line.

On *auscultation* loud pericardial friction rub is heard all over the dull area; a triple cantering rhythm is heard at the base.

No murmurs are heard over the region of the apex or at the base; the sounds are feeble.

Respiratory System.—The physical signs are: dulness and tubular breathing, with bronchophony over base of left lung posteriorly below spine of scapula. Right lung nothing abnormal.

Urine acid, sp. gr. 1020, no sugar or albumen; thick deposit of urates.

January 6th.—Not so well; had bad night; complains of pain in left shoulder. Temperature, 101·3. Pulse, 104. Respiration, 40.

The area of cardiac dulness extends upwards to upper border of second rib; to the right, to 2 f. b. to right of sternum; to left, half an inch to left of vertical nipple line.

A pericardial friction rub is heard over the base of the heart and a faint rub over tricuspid area; no rub is heard at apex or to right of sternum except over second right intercostal space.

As the area of cardiac dulness had increased considerably and the friction rub had diminished, and the man was in great distress, it was thought that there

might be pericardial effusion, so aspiration of the pericardium was performed. The needle was passed in through the fifth space close to the sternum and gradually pushed on, aspiration being tried at intervals, but no fluid was withdrawn, till at length it penetrated one of the cardiac cavities and some blood was withdrawn.

The operation was done without an anæsthetic, and the patient did not find the pain very severe.

January 17th.—Patient seems better; has had a good night. Pulse, 100. Respiration, 32. Temperature, 101·6.

Area of cardiac dulness extends upwards to upper border of second rib; inwards to 3 f. b. to right of sternum, to within ½ inch of right nipple; outwards 1 inch beyond nipple line. Hence it has increased somewhat since yesterday. No pericardial friction rub is to be heard.

January 18th.—Patient is improving; has slept well; much less pain in præcordium; area of cardiac dulness about the same.

January 19th.—Rather better. Pulse, 92. Respiration, 24. Friction rub is audible down right margin of sternum.

January 20th.—Still improving; area of cardiac dulness rather less; apex beat perceptible; friction rub still heard to right of sternum.

The patient henceforth made a steady recovery; the area of cardiac dulness gradually decreased and the heart sounds and pulse improved in character; the patch of pneumonia at the left base also cleared up.

On February 26th he got up for the first time.

On February 15th the note as to the extent of cardiac dulness was—it extends inwards to mid-sternal line, outwards to ½ inch outside vertical nipple line, upwards to third rib. The apex beat very feeble in fifth space just outside vertical nipple line; first sound rather weak; no murmurs.

He was discharged from the hospital on March 5th, when he could walk about and go upstairs without being in any way the worse for it.

Commentary.

This case is quoted to show how extensive the increase in the area of cardiac dulness may be in a case of pericarditis, owing to dilatation of the heart; it was thought that this increase might be due to fluid in the pericardial cavity, but aspiration of the pericardium proved that this was not the case, so that it could only have been due to increase in the size of the heart, that is, to dilatation of the heart.

That the needle was pushed in sufficiently far was proved by the withdrawal of blood eventually from one of the cavities of the heart; at one time the heart could be felt rubbing against the point of the needle in the course of the operation, yet no fluid could be withdrawn then.

Before the patient left the hospital the area of cardiac dulness had diminished considerably again and was almost normal in extent. It is evident therefore that in this case the heart recovered from its condition of dilatation, when the inflammation had subsided. Presumably the damage to the muscular

fibres was not severe, and the pericardium did not become adherent. Had the pericardium become adherent it is probable that the heart would not have again so nearly recovered its normal size, but would have been prevented from so doing by adhesions.

When the patient left the hospital there were no physical signs present to give rise to any suspicion of adherent pericardium, and he felt very well himself, and was able to walk upstairs without feeling any inconvenience.

Case C.

Case of Chorea and Pericarditis which proved rapidly fatal.

ALFRED H., æt. 8.

Admitted to Children's Hospital, Great Ormond Street, April 28, 1894. Died May 5, 1894.

History.—On April 13th, complained of pain in stomach, and was said to have 'inflammation of bowels.' He got better of this, and a week later was frightened by his father coming home drunk on April 20th. Next day he began to have choreic movements—on April 23rd he lost his power of articulation. No previous history of rheumatism or chorea.

Condition on Admission.—Anæmic, thin boy; frightened expression; understands what is said, but makes inarticulate grunts in reply; choreic movements marked. Pulse, 160. Respiration, 44.

Lungs.—Nothing abnormal.

Heart.—Apex beat fifth space ½ inch inside nipple line.

Cardiac dulness extends to upper border of third rib, and inwards to mid line of sternum. Rough systolic murmur at apex.

Next day after admission, on April 29th, pericardial friction rub heard at apex.

April 30th.—Friction rub heard at third left costal cartilage. Liver edge is felt two fingers' breadth below costal margin.

May 3rd.—Pericardial rub more distinct, and heard over large area.

May 4th.—Pericardial rub audible over most of the area of cardiac dulness, and also to the right of the sternum for a short distance.

Area of cardiac dulness much increased; extends 1 f. b. to right of sternum.

May 5th.—Much worse; great dyspnœa; severe attack of hæmoptysis. Dulness and bronchial breathing over front and upper half of back of right side of chest. Vocal resonance and fremitus increased over dull area.

Triple cantering rhythm and pericardial rub all over area of cardiac dulness.

Died 8.40 p.m.

Autopsy.

The pericardium occupied a large area uncovered by lung, and was injected; it was slightly more adherent than normal to chest wall. On opening the

pericardial sac there was evidence of recent pericarditis, and the pericardium was adherent over the interventricular septum to the heart. There was no excess of fluid in the pericardial cavity.

Heart.—Weight 7½ ozs., of considerable size; all its cavities, especially the right ventricle, much dilated; walls of right ventricle extremely thin and its cavity much enlarged.

Tricuspid orifice admits 2½ fingers; valves covered by fine recent vegetations.

Mitral orifice admits two fingers; auricular surface of valves present numerous fine recent vegetations.

On the aortic valves are some fine vegetations, but they are not much damaged.

Pulmonary valves normal.

Lungs.—Right lung dark and congested; firm and solid; portions sink in water.

Commentary.

This case shows how considerably the heart may be dilated in pericarditis, and how especially the right ventricle suffers, being the most thin-walled of the two ventricles. The increased area of cardiac dulness during life was proved by the autopsy to be due, not to fluid in the pericardium, but to dilatation of the heart, and especially of the right ventricle.

The condition of the right lung, which doubtless accelerated death, was curious, and seemed to be the result of extensive thrombosis.

This is also a case of great interest, from the fact

that the chorea directly followed on a severe fright, and was soon further complicated by an attack of endocarditis and pericarditis, which proved fatal.

Case D.

Case of Pericarditis in which death occurred just after formation of universal pericardial adhesions.

DAISY B., æt. 8.

Admitted to Children's Hospital, Great Ormond Street, October 21, 1893. Died, December 1, 1893.

History.—Taken ill ten days ago with pain in joints. Four months ago had scarlet fever. Has complained of pain in left side since; lately has fainted several times.

Condition on Admission.—Well nourished child. Temperature, 100. Pulse, 108. Respiration, 28.

Heart.—Impulse visible in fourth and fifth spaces, on palpation apex beat felt in fifth space in vertical nipple line. No thrill felt.

Cardiac dulness extends above to lower border of third rib; inwards to mid-sternum; outwards to nipple line.

On *auscultation*, blowing systolic murmur is heard at apex, which is not well conducted into axilla. Heart beat irregular; occasionally a beat is dropped.

Lungs.—Nothing abnormal. No swelling of joints.

Urine.—Acid. No albumen.

October 26th.—Erythematous rash of rheumatic character all over chest.

October 28th.—Heart action markedly irregular.

Temperature, 100. Pulse, 120. Respiration, 30. The irregularity of heart action was marked and persistent till November 4th.

On November 3rd a presystolic and systolic murmur were heard at apex.

November 7th.—Heart action regular; impulse more forcible, long distinct presystolic and systolic murmurs. Temperature, 99·8. Pulse, 130. Respiration, 36.

November 10th.—Thrill felt in second left intercostal space; heart still regular; area of cardiac dulness the same as on admission.

November 17th.—Heart action irregular again; more rapid. Pulse, 150. Respiration, 44. Temperature, 99·8. Wavy impulse visible in third and fourth spaces.

Area of cardiac dulness increased, extends outwards one finger's breadth beyond nipple line; inwards just beyond mid line of sternum; upwards to third rib. Child not so well; restless at night, wakes up screaming. Rumbling presystolic and systolic murmur at apex.

November 27th.—Child is worse. Pulse more rapid, 170. Temperature, 100. Respiration, 40. Area of cardiac dulness increased, extends to right border of sternum inwards; above to second rib; outwards 1 f. b. beyond nipple line. Pericardial rub audible in third and fourth spaces to left of sternum.

November 28th.—Child worse; pericardial tenderness and pain marked; rub more extensive. Pulse, 160. Temperature, 101·5. Respiration, 52. Pulse irregular in force and frequency.

November 30th.—Condition worse. Pulse, 140, irregular. Respiration, 48. Pericardial rub all over dull area; percussion not practicable owing to præcordial tenderness and to effect of blisters.

During night child became much worse and had several attacks of vomiting, one of which caused a syncopal attack, and she died at 4 a.m. on December 1st.

Autopsy.

On removing sternum heart was seen to be enlarged, reaching to right of sternum; a needle placed in fifth interspace to right of sternum entered right auricle; one in fifth space in nipple line pierced apex of heart.

Pericardium was universally adherent to heart by recent adhesions, which were easily broken down by finger except over area, half an inch in diameter, just inside apex, where adhesion was tough and of older standing. There was no fluid in the pericardial cavity.

Heart.—Much enlarged; cavities dilated; weight 9½ ozs.

Tricuspid orifice admits three fingers; is free from vegetations.

Mitral orifice admits two fingers, no stenosis of orifice; vegetations along auricular surface of valve, edge of which is slightly thickened; chordæ tendineæ a little shortened.

Aortic Valve.—Few fine vegetations on ventricular

surface. Valves probably were functional, but were not tested.

Lungs and other viscera normal.

Commentary.

This case illustrates the point that the increase of area of cardiac dulness during the attack of pericarditis was due, not to effusion of fluid, but to dilatation of heart. Further, the pericardium was at the autopsy found to be adherent to the dilated and enlarged heart, and no fluid was found in the pericardial sac.

The history of the illness is also instructive. For some weeks previous to the appearance of the pericardial rub there was evidence of cardiac mischief, as shown by the irregularity of pulse and the presence of murmurs which changed from day to day; the temperature was also slightly raised, and the child was very restless, and often woke up frightened at night without any apparent cause. The changing murmurs indicated the presence of active endocarditis, and the increase in the area of cardiac dulness was presumably due to myocarditis. It is possible that the inflammatory process began with endocarditis and myocarditis, and ended up with pericarditis. It seems, however, more probable that the pericarditis may have begun on the posterior aspect of the heart, so that the characteristic friction rub was not audible till the inflammation had extended round to the lateral and anterior surfaces; the fact that the friction rub was first heard at the apex and not at

the base, would tend to support this theory. Frequently in cases of pericarditis there are indications that the heart is affected some time before the pericardial rub is audible; namely, frequency and irregularity of pulse, increase of the area of cardiac dulness, etc., and it seems probable, therefore, either that myocarditis precedes the pericarditis, or that the pericarditis begins on the posterior surface of the heart, and ultimately extends round to the anterior surface in such cases.

Case E.

Case of Pericarditis in which the formation of Pericardial Adhesions, and their organization into fibrous tissue, took place while the patient was in Hospital.

HENRY D., æt. 9.

Admitted to Children's Hospital, Great Ormond Street, December 11, 1893. Died April 21, 1894.

History.—Taken ill with pains in joints fourteen days ago, since then he has been in bed; he complained of pain in left side of chest a week ago. No vomiting.

Condition on Admission.—Pale, thin, listless, perspiring over forehead, slight cough, no nodules. Pulse, 104. Respiration, 44. Temperature 102 on admission, 99° next morning.

Heart.—Diffuse, pulsation seen over third, fourth, and fifth spaces. Apex beat seen and felt in fifth

space in nipple line. The area of cardiac dulness extends above to second space; inwards to right margin of sternum.

A blowing systolic murmur is heard at the apex; the second sound is reduplicated occasionally at the base; the second sounds are clear.

Abdomen.—Liver dulness extends down to two fingers' breadth below costal margin, where the edge can be felt.

Lungs.—On percussion the note is impaired at both bases, over which crepitations are also heard.

Urine.—Sp. gr. 1020; trace of albumen.

December 14*th.*—Has had some hallucinations last two days; said he saw his father standing by the door and at window, etc.; wanders at times in his talk.

December 16*th.*—Pericardial rub heard down left border of sternum.

December 18*th.*—Well marked pericardial rub heard along sternum; area of cardiac dulness increased. Extends above to second space; inwards to one finger's breadth to right of sternum; left limit is in nipple line; tenderness over præcordial area; heart beating regularly. Pulse, 124. Respiration, 48. Temperature, 101.

December 19*th.*—Cardiac dulness increased to left, extends $1\frac{1}{2}$ f. b. outside nipple line; pericardial rub heard to right of sternum.

On the 22nd no friction sounds were audible, and on the 26th the area of cardiac dulness had diminished a good deal, extending only to mid line of sternum to right; but the boy was very lethargic, and had on the 23rd and 24th passed motions into the bed.

After this he improved slowly, but steadily, and on February 3rd the area of cardiac dulness was almost normal, extending upwards to third rib; inwards to left edge of sternum; outwards to nipple line. There was diffuse pulsation over third, fourth, and fifth spaces to left of sternum; the last note as to the position of the apex beat, was that it is in fifth space half an inch outside nipple line. At apex systolic and short diastolic murmurs were audible; no murmurs at base.

On February 15th he was discharged, much improved, to Convalescent Home at Highgate. Here he remained pretty well till February 28th, when he vomited several times; March 6th he complained of pain in right side; and on March 7th friction sounds were heard at right base in axillary line.

On March 15th he was readmitted to the hospital; his condition then was as follows:

Child is anæmic; there is slight general œdema, most marked in feet; has orthopnœa. *Nodules* seen on both elbows and both knees.

Circulatory System.—Pulse, 134, regular; veins of neck prominent; carotid pulsation marked; epigastric pulsation.

Pulsation visible over greater part of left front, from second to sixth space. Apex beat in sixth space 2 f. b. outside nipple line.

Cardiac dulness extends upwards to third space; extends 1 f. b. to right of sternum; 2 f. b. outside nipple line.

There is a systolic murmur at apex conducted to axilla; also presystolic rumbling murmur.

Lungs.—Dulness at base of right lung up to middle

of scapula, with weak breathing and loss of voice, sounds over dull area.

Liver edge cannot be felt, owing to abdominal distention; felt next day, it extended down to the level of the umbilicus.

March 19*th.*—Œdema of feet considerable; general œdema of trunk of slight degree, more marked in lumbar region at base of spine, also perceptible over sternum.

Liver edge reaches below umbilicus.

Heart.—Area of dulness as before; diastolic murmur audible to left of sternum.

March 27th.—Œdema has diminished greatly, almost gone: but the boy is very drowsy and apathetic.

April 5th.—Œdema has returned; face puffy; irritable cough; crepitations over bases of both lungs; still some dulness at right base.

April 9th.—Purpuric rash has come out over nearly the whole body.

From this time the boy got steadily worse, and the œdema of the legs and subsequently of the trunk became very considerable. The liver extended down to below the level of the unbilicus. The pulsation in the third, fourth, and fifth left intercostal spaces, which on close examination seemed to be a systolic retraction, remained persistent and marked, and the area of cardiac dulness much enlarged till the end.

On April 21st he was much worse, and his pulse became very slow, weak, and irregular. He died at 10·45 that evening.

Post-mortem Examination.

Body very œdematous; extensive œdema of legs, scrotum, and trunk.

Pericardium.—There were adhesions between the pericardium and chest wall. The pericardium was universally adherent to the heart, which was seen to be much enlarged. The adhesions could with difficulty be broken down by the finger.

Heart.—Weight 10 oz. All its cavities were considerably dilated; there was not much hypertrophy.

Tricuspid orifice admitted two fingers easily; valves free from vegetations.

Pulmonary valves normal.

Mitral orifice admitted two fingers barely; there was no stenosis of the orifice. There were some vegetations along the edges of the valves.

Aortic valves competent and normal in appearance.

Lungs and Pleuræ.—There were firm adhesions between left pleura and chest wall, and in right pleural cavity there were about 8 oz. of clear fluid.

The right lung was collapsed and œdematous, its substance being firm.

Abdomen.—There was a small quantity of fluid in the peritoneal cavity.

The liver was very greatly enlarged, and weighed 48 ozs. It did not present a typical nutmeg appearance on section, but was rather pale, and its substance was tough. The hepatic veins were enormously dilated.

There were some adhesions on the lateral surface of liver.

Spleen and kidneys firm, but otherwise normal.

Commentary.

This case illustrates the dilatation of the heart in pericarditis; after the initial attack, however, the heart appeared to almost recover its normal size, except that the apex remained displaced outwards. It was the first attack of rheumatism, and presumably of pericarditis. From it the boy recovered sufficiently to go to a convalescent home, but after he had been there about a fortnight he had a fresh attack of rheumatism, as shown by the eruption of rheumatic nodules on his elbows and knees; he also had some pleurisy and probably fresh pericarditis, though when he came back to the hospital again no friction rub was to be heard,—the heart was then much dilated and there were signs of right ventricle failure, as shown by the enlarged liver and œdema of the lower extremities. Though he seemed at first to improve a little and the œdema diminished, the heart did not recover from its condition of dilatation, presumably owing to the adherent pericardium, and also to the effects of the damage done by the myocarditis to the muscular fibres. He got rapidly worse again, and the œdema of the legs increased and became universal; the liver also became enormously enlarged. At an early period there was œdema over the sternum and præcordial area, but as there was such general œdema it could hardly be of any significance with regard to

the adherence of the pericardium. At the autopsy, the pericardium was found to be universally adherent, and the heart much dilated. It is possible that the attack of pericarditis in December did not cause adherence of the pericardium—at any rate, universal adhesion,—as the heart almost recovered its normal size afterwards. The eruption of nodules and pleurisy which occurred while he was away signified a fresh attack of rheumatism, and it is probable there was also some fresh pericarditis then, which resulted in the universal adherence of the pericardium.

The right ventricle became dilated, as evidenced by the increase of the area of cardiac dulness, and soon symptoms of right ventricle failure set in, namely, enlargement of liver, œdema of lower extremities, etc. These symptoms continued to increase in severity in spite of rest in bed and the administration of cardiac tonics.

In the absence of evidence of any serious valvular lesion, and with a history of recent pericarditis, it was only natural to suppose that adherent pericardium was responsible for the symptoms, and had caused the break-down of the right ventricle by effectually embarrassing its movements, and also preventing the possibility of its recovery when once it had given way; and this diagnosis was confirmed by the autopsy.

Case F.

Case of Universally Adherent Pericardium which gave no definite physical signs of its existence during life.

EVELYN R., 16.

Admitted to St. Mary's Hospital, April 15, 1893. Died June 28, 1893. Admitted complaining of shortness of breath, and swelling of abdomen and feet.

History.—Was well till last Christmas, 3½ months ago.

Then she had an illness which kept her in bed two weeks. She remembers she had pains in her back, and was short of breath; was sick several times, and had headache. She also had some cough, but does not remember having any pain in her joints.

Since this illness she has not been well, but always short of breath, and her feet swell sometimes after walking.

She does not remember ever having had rheumatic fever or any other illness.

Condition on Admission.—Fairly well-nourished girl; pale face; no cyanosis; does not complain of pain; feet œdematous. Pulse, 112. Respiration, 24. Temperature, 97·5.

Cardio-vascular System.—Pulse, 112, regular in force and frequency; wave small and ill-sustained; artery small, easily compressible, can be felt between the pulse beats.

No pulsation of veins in neck or fulness of jugulars.

Heart.—Apex beat not seen or felt.

Area of cardiac dulness of normal extent.

On *auscultation*, heart sounds are best heard in fifth space, just inside the nipple line; the first sound is short and weak; a feeble second sound is also heard.

The sounds are equidistant, and somewhat like the ticking of a watch.

At the base, a weak aortic and a slightly louder pulmonary second sound is heard.

There is no pulsation visible in any intercostal space, and no pulsation in epigastrium.

The abdomen is uniformly distended, and is dull on percussion except for about three inches on either side of umbilicus. The dulness in the flanks varies with change of posture. Thrill indicating presence of free fluid in the abdomen is easily felt.

Liver is enlarged, and extends downwards halfway to umbilicus.

Right Lung and chest normal; there are signs of fluid in left pleural cavity, dulness, with loss of vocal fremitus and resonance below spine of scapula.

Urine.—Sp. gr., 1025; acid; no albumen; thick deposit of urates.

On April 20th, 48 ozs. of fluid were withdrawn from left pleural cavity.

After the aspiration, the patient expectorated a quantity of frothy pale yellow liquid, slightly blood-stained; 14 ozs. in all.

This fluid was found to contain albumen, and resembled serum in its character.

Presumably this was due to exudation of serum into the smaller air-passages, consequent on the sudden

relaxation and expansion of the lung on its release from pressure by the fluid, the expansion causing a sudden loss of mechanical support to the capillary walls, and perhaps inducing a negative pressure on some of the alveoli, both of which conditions would tend to produce a serous exudation.

On April 21st she felt better, and the œdema of feet had quite disappeared.

On April 30th, the abdomen being much distended by fluid, a Southey's tube was inserted, and ℥ 170 of clear fluid drawn off.

This fluid was of yellow colour, rich in albumen, and coagulated very soon.

The abdomen soon filled up again after tapping, and, on May 11th, ℥ 176 were withdrawn from the abdomen; on May 23rd, ℥ 180; on June 5th, ℥ 218; on June 15th, ℥ 106.

The patient seemed to make no progress, and became gradually more and more depressed in mind.

The circulation and condition of the heart did not improve, and the liver became gradually larger; but, till a few days before death, there was no œdema of feet and legs.

On June 25th she developed acute mania, screaming and laughing alternately, and talking incoherently and continuously.

She also used abusive and offensive language, and attempted to bite and scratch.

She was removed to the isolation wards, and died on June 28th—three days later—from asthenia and exhaustion, never becoming sensible again.

Post-mortem Examination.

Peritoneal cavity was nearly full of clear serous fluid.

On opening the thorax, the pericardium was seen to be universally adherent to the heart, but was not abnormally adherent to the chest wall; there were a few adhesions between the pericardium and the left lung.

The pericardium was about ¼ in. thick, and very tough, and could not be separated from the heart wall without tearing it.

Nearly the whole of the lower surface of the right ventricle was adherent to the diaphragm by firm organized adhesions.

The left ventricle was also surrounded by firm fibrous pericardium.

The heart was bound down to the diaphragm by the fibrous adherent pericardium, which seemed too tight and firm to allow of any cardiac dilatation or hypertrophy.

The heart was abnormally small, weighing only about 6 ozs.

None of its cavities were dilated, and the valves were all normal, and free from vegetations.

The mitral and tricuspid orifices were of normal size.

In the right pleural cavity there were about ℥ 20 of clear serous fluid.

Lungs congested; some grey miliary tubercles at the apex of the left lung.

Liver much enlarged; 'nutmeg' on section; no enlarged glands in hilum.

Brain and Meninges.—Nothing abnormal detected.

Peritoneum and intestines studded with small grey miliary tubercles.

Commentary.

The most striking features in the case were the constant re-accumulation of fluid in the peritoneal cavity; the feebleness of the pulse and its smallness, the absence of the apex beat, and the poor circulation generally.

There was nothing to indicate the presence of adherent pericardium, which was not suspected during life.

The area of cardiac dulness was normal; there was no retraction of spaces; the absence of apex beat, and the weak and muffled heart sounds, were the only physical signs which might have suggested a possibility of adherent pericardium.

The constant recurrence of ascites after frequent tapping was difficult to explain, and it was thought during life to be due possibly to cirrhosis of the liver, together with right ventricle failure. The presence of tubercle in the peritoneum found post-mortem might have accounted for the ascites; but it was not suspected during life, as there was no abdominal tenderness or tympanites, no intestinal disturbance, and the temperature was normal or subnormal throughout. During life there was nothing to give

rise to a suspicion of tubercular peritonitis. Post-mortem, the heart was found to be enveloped in a thick, firm mass of adherent pericardium, and was bound down to the diaphragm. It would seem that in this case the heart had not become dilated during the attack of pericarditis, in which the pericardium became adherent. Probably this had occurred in childhood, and the pericardial adhesions, becoming organized into firm fibrous tissue, had prevented the heart from developing and growing to its proper adult size; this would account for the poor circulation, the small and undeveloped heart being inadequate for the adult state.

Case G.

Case of Universally Adherent Pericardium in which the Right Auricle was obliterated by the adhesions.

FREDERICK B., labourer.

Admitted to St. Mary's Hospital, May 2, 1891. Died April 21, 1892.

Admitted for shortness of breath, and swelling of feet and legs.

History.—Was well till one year ago, when he had 'pleurisy' on both sides.

Since then he has had pain in the chest over the pericardial region, and shortness of breath. Whenever he walked, his feet and legs swelled.

No history of rheumatic fever or any other illness at any time could be obtained.

Condition of Admission.—Fairly well-nourished man; pale face; no cyanosis.

There is general œdema of legs and feet, and œdema of trunk on the side on which he lies.

Pulse, 104. Respiration, 28. Temperature, 98·4.

Cardio-vascular System.—Pulse 104, regular in force and frequency; easily compressible; wave small and ill-sustained; artery small; can be felt between the pulse beats.

Heart.—Apex beat cannot be seen or felt.

No pulsation observable in any of the intercostal spaces.

Percussion.—Cardiac dulness could not be accurately mapped out, owing to dulness at both bases.

Auscultation.—Sounds are best heard in fifth space just inside nipple line—a weak reduplicated first sound and a second sound.

There is no murmur to be heard at the apex.

At base, a weak aortic second sound is heard in second right space; reduplication of second sound is heard in second left space.

Lungs.—There is dulness over left back and front, and impaired vocal fremitus below the angle of the scapula, and on auscultation there is impaired entry of air and impaired vocal resonance.

There is some flattening and deficient movement of left side of chest.

There is impaired resonance over back and front of right lung at the base.

Liver.—Enlarged; it extends down to three fingers' breadth below costal margin. It does not pulsate.

There is some fluid in the peritoneal cavity.

Urine.—Sp. gr. 1024; no albumen.

June 25th.—Œdema of lower extremities and trunk still present.

Raising the foot of the bed by blocks caused œdema of legs to decrease somewhat, but œdema of thorax to increase, and the face to become more puffy.

Condition of heart about the same.

July 6th.—Southey's tubes were inserted into both legs, and, during the next four days, ℥ 261 of fluid escaped from them.

During July and August considerable quantities of fluid escaped from the legs, and the œdema diminished somewhat.

September 2nd.—There is still considerable œdema of legs and trunk.

Patient has a pale waxy appearance, and looks anæmic.

Heart.—Apex beat can just be felt in fifth space, 1 inch below and ½ inch internal to nipple. It is extremely feeble. There is no epigastric pulsation.

On *auscultation*, weak reduplicated first sound and weak second sound heard at apex; no murmurs are present.

There is reduplication of the second sound at the pulmonary cartilage.

During September, ℥ 1276 of fluid, that could be measured, escaped from the legs; in first half of October, ℥ 652; during November, ℥ 1307; from December 1st–15th, ℥ 597.

The fluid that escaped was of pale yellow colour, sp. gr. 1015, alkaline, and contained 0·07 per cent. of albumen and various salts.

In spite of this, the œdema of the trunk and lower extremities persisted, and nothing seemed to make any impression on it. The patient gradually got weaker, and during February, 1892, signs of fluid in right base developed, which afterwards became purulent. He died on April 21, 1892.

Post-mortem Examination.

Body of a man with very great œdema of lower extremities and trunk.

On opening the thorax, the pericardium was found to be universally adherent to the heart, but did not appear to be abnormally adherent to the chest wall.

The pericardium was much thickened, and the adhesions were of a firm fibrous nature, and could not be broken down by the finger.

The right auricle was found to be almost entirely obliterated by the thick adherent pericardium, which had so compressed it that the orifices of the vena cava superior and inferior were almost in direct contact, and the opposing currents of blood must have met just over the tricuspid orifice, there being practically no auricular cavity.

The inferior vena cava was enormously dilated.

The tricuspid orifice was dilated, and admitted four fingers.

The tricuspid valves were free from any vegetation.

The right ventricle was somewhat dilated, and hypertrophied.

The left auricle was of normal size.

The left ventricle was not enlarged.

The mitral and aortic valves were normal.

The right pleural cavity contained about 1½ litres of pus.

Both **lungs** were adherent to the chest wall at the bases by thick fibrous adhesions.

Liver.—Much enlarged; 'nutmeg' in character on section.

Kidneys normal in appearance.

Commentary.

This case explains and illustrates what is meant by the statement, 'That the excessive severity of the symptoms, out of proportion to and unexplained by the physical signs, is of great importance in the diagnosis of adherent pericardium.'

The diagnosis of adherent pericardium was in this case made during life on the following grounds:—

The most marked feature of the case was the extreme and general dropsy, which did not yield to rest, or treatment of any kind.

There was no evidence of kidney disease, which might have given rise to the œdema.

The full jugular veins, the enlarged liver and epigastric pulsation, together with the œdema, showed that there was serious obstruction to the venous return to the right auricle—presumably from dilatation of right ventricle, and tricuspid regurgitation. (The autopsy revealed the fact that there was a further contributory cause of the obstruction to the venous return, namely, the practical obliteration of the right auricular cavity by the pericardial adhesions.)

There was no pulmonary disease, such as chronic bronchitis or phthisis, which could have caused the break-down of the right ventricle.

There was no evidence of any valvular disease of the left side of the heart—aortic or mitral—which might secondarily have caused embarrassment of the right side of the heart by back-working, *i.e.* obstruction to the entry of blood from the lungs into the left auricle, caused by mitral reflux. The absence of the apex beat at first, and its extreme feebleness when it was felt, as well as its position in the fifth space 1½ inches internal to nipple, together with the small weak pulse, showed that the left ventricle was not hypertrophied, but was acting feebly, and was embarrassed. It was obvious that the œdema was of cardiac origin, and it was concluded that an adherent pericardium was present, and so hampered the right side of the heart as to render it incompetent, while it interfered to a less degree with the thicker-walled, more resistant left ventricle.

In this case, however, the pericardial adhesions produced the remarkable result of the obliteration of the cavity of the right auricle, so that the vena cava superior and inferior practically emptied into each other, so to speak, and the current of blood in each was dammed back by the other. This had operated greatly to the disadvantage of the inferior vena cava, which was very much dilated. It would also explain the fact that, when the legs were raised, the œdema in neck, face, and arms became very much more marked, as then the current of blood from the vena cava inferior would have the advantage of gravity.

There was at no time any cyanosis, and no dyspnœa while the patient was at rest in bed, and no indications of pulmonary congestion. This seemed remarkable when the other symptoms were so severe; and this alone was sufficient to give rise to a suspicion of adherent pericardium, for it showed that it was not back pressure or obstruction to the flow of blood through the lungs which had caused the symptoms of right ventricle failure, but something which directly embarrassed the right side of the heart, and hindered the venous return to the right auricle either by direct obstruction or by reflux through the tricuspid orifice.

Case H.

Case in which a diagnosis of Adherent Pericardium was made in life, but which did not terminate fatally, so that the diagnosis was not verified.

ELIZA D., æt. 50.

Admitted April 25, 1893. Discharged June 15, 1893.

Admitted for pain over præcordial region and œdema of legs.

History.—About three years ago she first had pains in her chest, and some swelling of the legs. Twenty-eight years ago she said she had rheumatic fever. She is a laundress by occupation. She has

seven children, and says she always had to lay up during the later months of pregnancy, owing to shortness of breath.

Condition on Admission.—No dyspnœa or cough; sallow complexion; injected capillaries on face; no cyanosis. Pulse, 80. Respiration, 24. Temperature, 97·8.

Cardio-vascular System.—Pulse irregular; wave small and compressible; artery small. External jugular vein on left side is distended, fills from below, and pulsates.

Heart.—Apex beat not seen or felt.

There is marked epigastric pulsation.

There are marks of leech-bites over the præcordium, due, she said, to leeches applied when she had rheumatic fever.

On *palpation*, a diffuse and forcible right-ventricle impulse is felt; there is no thrill. The apex beat is not perceptible.

The *area of cardiac dulness* extends upwards to third rib; inwards to left border of sternum; outwards 1½ inches beyond nipple line.

On *auscultation*, short sharp first sound, closely followed by second sound, is heard over region of apex. Over the tricuspid area a loud systolic murmur is heard, replacing the first sound. This becomes less distinct as the stethoscope is carried outwards, and is lost as soon as the vertical nipple line is reached. No murmurs are audible at the base.

The liver extends down to halfway between the costal margin and the umbilicus, and pulsates. Legs œdematous. No ascites.

Lungs.—Resonance and entry of air good; no abnormal sounds; no dyspnœa; slight cough.

Urine.—Sp. gr., 1020. Trace of albumen; high-coloured deposit of urates.

May 11*th.*—Patient is much better. Liver margin is felt only two inches below ribs; it pulsates slightly; œdema of legs is much diminished.

The apex beat is still not visible or palpable; the same systolic murmur is still audible. The pulse is still irregular.

May 18*th.*—Improving. Patient gets up; no œdema of legs.

Liver does not pulsate, but still reaches 1½ inches below costal margin. Epigastric pulsation not so marked. Apex beat is still not to be seen or felt; the heart sounds are best heard in fifth space. Still some pulsation in left jugular vein.

May 23*rd.*—Better. The apex beat can just be felt in fifth space 1½ inches outside vertical nipple line.

Pulse, 66. Still irregular.

June 15*th.*—Patient much better; liver only just felt below ribs, and does not pulsate. Pulse still irregular and small.

Apex beat perceptible in fifth space just outside vertical nipple line. Sounds much the same.

The systolic murmur is most marked over the tricuspid area; it is lost at the apex.

Patient discharged June 15th, relieved.

Commentary.

In this case the most marked features were the œdema of the legs and the enlarged liver, evidencing obstruction to the venous return due to tricuspid regurgitation, as proved by the pulsation of the liver and veins of the neck.

There was no respiratory distress, and no dyspnœa, the respirations being normal; nor was there any evidence of pulmonary congestion from obstruction to the return of blood to the left auricle; nor any lung trouble, such as chronic bronchitis, which might have given rise to right-ventricle failure.

There was a systolic murmur, best heard over the tricuspid area, and not heard at the apex, or conducted to the axilla.

There was no evidence of valvular disease of the left side of the heart, or of hypertrophy of the left ventricle; indeed, the apex beat was not to be felt, while the epigastric pulsation of the right ventricle was very noticeable. This fact alone would raise a suspicion of the presence of adherent pericardium, and, taken together with the break-down of the right ventricle, for which there was no valvular disease of the left side of the heart or pulmonary trouble to account, it was concluded that it was an adherent pericardium which was causing this embarrassment of the right heart.

The presence of leech-bites over the pericardium was also strong evidence of this, as it is improbable that leeches would be applied over that region for any other disease than pericarditis.

There was, of course, the possibility that the tricuspid valve itself might have been damaged by endocarditis and rendered incompetent, but if that had been the case the patient would probably have had symptoms at a much earlier period. Again, endocarditis affecting the right side of the heart, and especially the right side alone, is so rare, that the probabilities were greatly in favour of adherent pericardium.

This case would also illustrate the point that it is the thin-walled right ventricle fixed down to the diaphragm which suffers most as a result of adherent pericardium.

Case J.

Case of Adherent Pericardium of recent date.

ELEANOR T., æt. 9.

Admitted to Children's Hospital, Great Ormond Street, December 11, 1893. Died February 1st, 1894.

Admitted for pain in chest, and shortness of breath.

History.—Had scarlet fever at the age of six, chorea at the age of eight.

No history of any attack of rheumatic fever, but she has complained at times of pains in joints.

No history of pericarditis.

The child has had a cough for three months, and complains of pain in left shoulder; has had to be propped up with pillows in bed, owing to dyspnœa: the face is pale; there is no cyanosis.

Family History.—Father and mother alive and well; no history of rheumatism. Three children died before the age of twelve, of measles and whooping cough. One died at the age of seventeen from chest trouble.

Condition on Admission. — Somewhat thin child; pale face; red eyelids; nose depressed.

No swelling of joints; some pain in left shoulder.

There are nodules on the outer margin of both patellæ, on the condylar ridge of both femora, on both olecranon processes, and on the backs of both hands.

Pulse, 112. Respiration, 32. Temperature, 100.

Circulatory System. — Pulsation of carotids visible; some fulness of veins of neck.

Pulse, 120; regular in force and frequency; easily compressible; wave somewhat sudden.

Heart.—Apex beat visible in sixth space, one inch outside vertical nipple line; epigastric pulsation well marked.

Cardiac dulness extends to upper border of third rib above; to right border of sternum internally; and one inch outside nipple line to the left.

Apex beat. There is a fair thrust, somewhat diffuse; a presystolic thrill is felt over area of apex beat.

At the apex a rumbling presystolic murmur and a blowing systolic murmur are heard; also a low-pitched first sound and a weak second sound; the systolic murmur is well heard in the axilla and at the angle of the left scapula.

The pulmonary second sound is heard over the aortic area, and is accentuated; the aortic second sound is feeble; there are no murmurs at the base.

Respiratory System.—Respiration, 38. There is some dyspnœa, the alæ nasi working a little; the movements of the chest wall are equal on both sides.

No dulness on percussion: scattered rhonchi heard over both lungs, and some crepitations at the left base and in the left axilla.

Liver not felt below costal margin.

Tongue clean; appetite fair.

Urine.—Acid; sp. gr. 1026; no albumen; thick deposit of urates.

December 15th.—Eruption of rheumatic erythema of type of erythema marginatum over chest and arms.

December 20th.—Rash has all faded.

Temperature, 100·4, evening. 99·2, morning.

December 28th. Fresh eruption of the rheumatic erythema all over the body and limbs. Temperature, 100.

Heart.—No change. Pulse, 104. Respiration, 32.

December 30th.—Rash has all faded again, and the skin is quite clear.

January 1st.—Temperature rose to 102·4, and varied between that and 99·4 till January 8th, when it came down to 98·4.

On January 2nd, crepitations were heard at the left apex, and in the left axilla, and the thrill over the præcordial region was more marked.

No friction sound was heard, and there was no increase of cardiac dulness. Temperature, 102·6. Pulse, 148. Respiration, 44.

No tenderness over præcordium.

January 5th.—Liver can be felt two fingers' breadth below costal margin, it is tender on palpation.

January 7th.—The nodules on both patellæ have become compound from the eruption of fresh ones.

January 10th.—Fresh nodules on malleoli of both legs: those on the elbow are larger than on admission.

January 11th–23rd. Patient seems a little better; still very pale; troublesome cough.

January 24th.—Not so well; appetite bad. Pulse, 140. Respiration, 44. Temperature, 100. Grunting respiration; liver enlarged and tender; no œdema of legs.

January 25th.—Pulsation of carotids more marked; pulse wave sudden and ill-sustained, of water-hammer character. Diastolic murmur can be heard at aortic cartilage accompanying second sound.

Murmurs at apex much the same; no friction rub.

January 31st.—Child has been getting gradually worse.

Cardiac impulse more marked; the apex beat is diffuse and is felt in the axilla in the sixth space.

Epigastric pulsation well marked.

Liver tender; its edge can be felt one inch below the level of the umbilicus; it is not pulsating.

The jugular veins pulsate.

Diarrhœa troublesome.

February 1st.—There is œdema of both feet, and the child is worse. She died at 3.45 p.m.

Post-mortem Examination.

Chest.—On opening the thorax the heart was seen to be much enlarged.

The pericardium was not abnormally adherent to

the chest wall, but was universally adherent to the heart by adhesions which could be broken down fairly easily, and in the meshes of which some fluid was enclosed.

Heart.—The heart was enlarged, and seen to be everywhere coated with organized lymph of some standing after the stripping off of the pericardium, which could be torn off by the fingers.

Right ventricle dilated and hypertrophied. Tricuspid orifice admitted two fingers. Valve normal. Pulmonary valve normal.

Mitral orifice admitted tips of two fingers; there was some thickening of the valve; chordæ tendineæ normal. Left ventricle hypertrophied and somewhat dilated.

Aortic Valves.—Small vegetations on anterior semi-lunar valve, edges of valves somewhat dragged down; from the appearance of the valve, one would not expect much regurgitation.

Abdomen.—Some fluid in peritoneal cavity.

Liver.—Weight 26 ozs.; much enlarged; 'nutmeg' on section.

Spleen.—Slightly enlarged; firm.

Kidneys.—Slightly enlarged and firm; capsule did not peel well, being adherent in places.

No microscopic change on section.

Commentary.

In this case the child was not greatly distressed on admission to the hospital, there being no œdema of

limbs or enlargement of liver; the chief trouble was dyspnœa.

The heart was greatly enlarged, the apex being in the sixth space outside nipple line, and the apex beat was diffuse though fairly forcible; the systolic murmur pointed to mitral regurgitation, but the murmur did not entirely replace the first sound, which was rather sharp in character, showing that the regurgitation was not very severe. The mitral regurgitation would not account for the downward displacement of the apex beat, which evidenced considerable enlargement of the left ventricle; the forcible apex beat and the fair-sized regular pulse showed there was some hypertrophy as well as dilatation.

There was no evidence of aortic disease on admission, or of kidney trouble which might account for this hypertrophy: it was probable, therefore, that there was some other cause, such as adherent pericardium, to account for this cardiac enlargement, resulting from an antecedent attack of pericarditis, in which the heart had dilated considerably: the pericardium had then become adherent to the heart, before it had recovered its normal size; subsequently some hypertrophy had taken place.

There was no history of a previous attack of pericarditis, hence the diagnosis of adherent pericardium was the more difficult to make with certainty.

The engorgement of the liver and the epigastric pulsation showed that the right ventricle was in trouble more than would be expected from the amount of mitral regurgitation present.

This did not improve with rest in bed and treatment, as would be expected; the presystolic murmur was of an indistinct rumbling character, and not of the rough, blowing character which leads up to a short, sharp first sound, and is indicative of mitral stenosis, which is rare in young children.

The pulse, position of apex beat, character of murmur, were all against mitral stenosis of any severity. This being so, and pulmonary disease being excluded, it seemed probable that adherent pericardium was the cause of the serious embarrassment of the right side of the heart.

The child went steadily down hill while in hospital.

The presence of numerous nodules on admission, and the eruption of fresh ones while in hospital, was (as Dr. Cheadle states in his book on the 'Rheumatic Affections of Children') of grave prognostic significance, as well as the eruption of the rheumatic erythema and the irregular temperature: they indicated that the rheumatism had gained firm hold on the child, and was still active. The rise of temperature on January 1st to 102, following on the eruption of the rash a few days before, probably marked the commencement of a fresh attack of peri- and endocarditis; and the progressive enlargement of the liver, noted first on January 5th, with the increase of respiratory distress, showed that the heart was beginning to fail, and that compensation had broken down completely. The appearance of the aortic diastolic murmur, on January 25th, was a further and significant indication of the presence of active endocarditis.

Whether she would have recovered from the acute

endocarditis, had her heart been unembarrassed by adherent pericardium, it is impossible to say; but, hampered as it was by the adherent pericardium and already considerably dilated, the fresh attack of endocarditis soon proved fatal.

The post-mortem examination showed that the valves were very slightly affected, and that the most serious lesion was the adherent pericardium.

The 'nutmeg' liver indicated that there must have been back-working and hepatic congestion for some considerable time, due to the inefficient action of the right ventricle and consequent obstruction to the venous return.

Case K.

Case of Adherent Pericardium of old standing, complicated by severe valvular lesions.

ELIZA F., æt. 32; laundress.

Admitted to St. Mary's Hospital, March 18, 1893. Died June 15, 1893. Admitted for shortness of breath, swelling of feet and legs after standing, and pain over præcordial region.

History.—Has had several attacks of rheumatism, the first being at the age of six, and the last three years ago.

In December last, four months ago, she noticed that her abdomen was swollen and her feet swelled after standing.

She has had several attacks of palpitation and pain in her chest, and has been gradually getting worse lately.

Condition on Admission.—Poorly nourished, with sallow complexion. She lies in bed on her back, and breathes comfortably without being propped up. Pulse, 57. Respiration, 27. Temperature, 98·5.

Cardio-vascular System. — Pulse slow, 57; irregular in force and frequency; artery small, easily compressible, and felt between beats.

A distinct double impulse is felt by one finger at height of systole, which is well shown by sphygmographic tracing: a typical 'bis feriens' pulse.

Heart.—Apex beat visible in seventh space, ¾ inch outside vertical nipple line. A wave of pulsation is visible in the second, third, and fourth intercostal spaces to the left of the sternum. The spaces appear to be retracted during systole. The apex beat does not change its position during deep inspiration. There is well-marked epigastric pulsation, and the lower part of sternum is lifted by the forcible impulse of the right ventricle; while the left half of the epigastrium, just inside the lower intercostal spaces, is pulled in during systole, and returns to its former position during diastole.

The apex beat on palpation is felt as a forcible thrust over a considerable area. There is no thrill.

Cardiac dulness extends upwards to second intercostal space, and inwards to 1 inch to the right of sternum.

On *auscultation*, at apex a loud harsh systolic murmur is heard, which is conducted into the axilla.

It does not entirely replace the first sound, which is low pitched and prolonged in character. A faint diastolic murmur is also heard at the apex, and a reduplicated second sound.

At *aortic cartilage* a rough systolic murmur and a diastolic murmur are heard; the diastolic murmur is well heard over the sternum.

Over *tricuspid* area a systolic murmur is heard.

The abdomen is rather distended; the left half of the epigastrium moves very little in respiration.

The liver extends down to $\frac{3}{4}$ inch above umbilicus; it is not tender, and does not pulsate.

The veins in the neck are full, but are not pulsating.

Pulsation is noticed in veins on dorsum of hand.

There is no œdema of the legs or feet.

Lungs.—Resonant on percussion over back and front. No adventitious sounds heard.

Urine.—Sp. gr. 1020. No albumen.

Such was the condition on admission. During the first fortnight in hospital the patient improved, but she then had several attacks of vomiting, and on April 10th pulsation of the liver and of the left external jugular was first noticed, evidencing severe tricuspid regurgitation.

Soon after this, it was noticed that there was marked pulsation of a superficial vein to the left of the sternum. On careful observation, it was seen that the vein filled during diastole, and emptied during systole.

She got gradually worse, and fluid began to accumulate in the peritoneal cavity.

On May 9th, the abdomen was tapped, and ℥ 210 of fluid was removed.

Fluid soon reaccumulated after tapping; and at short intervals, in four tappings, ℥ 698 of fluid were removed from the abdominal cavity.

She became gradually weaker and much emaciated, and died on June 15th.

Post-mortem Examination.

The pericardium was found to be universally adherent by old and tough adhesions to the heart, and to the chest wall in front over an extensive area, from the second left space to the apex in the seventh space, and to the posterior surface of the sternum as far up as the second rib.

The right ventricle was entirely bound down to the chest wall in front and the diaphragm below.

The left ventricle was also attached by firm adhesions to the chest wall at its anterior border and apex, where it came in contact with it.

Where it was overlapped by lung in front, it was intimately adherent to the thin layer of collapsed lung, which, again, was adherent to the anterior chest wall.

NOTE.—In order to observe the adhesions between the pericardium and chest wall, the heart and pericardium were removed with the portion of chest wall excised for the purpose of making the examination. If the sternum is removed and the pericardium detached from it before removal, the true extent and nature of

the adhesions between the pericardium and chest wall cannot be accurately observed.

Heart.—Of great size. Weight 18 ozs.

Left Ventricle.—Much hypertrophied and dilated. Mitral orifice admits one finger only. The orifice is 'button-hole' in shape, and the valves rigid, contracted, and adherent at their margins.

Right Ventricle.—Very small cavity, only about half the size of that of the left ventricle, but the walls are very thick and are much hypertrophied.

Tricuspid orifice admitted only two finger tips.

The valves were adherent all along their margins, and were much thickened; the orifice was funnel-shaped.

Aortic Valves.—Orifice much stenosed, about two-thirds occluded. The valves were rigid, thickened and adherent at margins.

Pulmonary Valves.—Normal.

Liver.—Much enlarged; 'nutmeg' on section.

Kidneys.—Firm; normal appearance on section.

Lungs.—Congested and œdematous.

Commentary.

This was a remarkable case during life, and the autopsy also revealed remarkable cardiac lesions, some of which were not suspected during life. It is extraordinary that, with such serious lesions of every valve in the heart except the pulmonary, complicated by adherent pericardium, the patient should have lived to the age of thirty-two. The most distressing symptom during the time she was in hospital was

the constantly recurring ascites, necessitating frequent tapping of the abdomen. There was at no time any œdema except at first after standing; there was no great dyspnœa till a week or two before death. She complained frequently of pain in the præcordial region, and also of feeling faint; at one time she had frequent attacks of vomiting. These, briefly, were the chief symptoms. With regard to the physical signs, the most striking feature was great hypertrophy of the heart, and especially of the left ventricle.

The diastolic murmur heard at the aortic cartilage and down the left of the sternum, implied the presence of some aortic regurgitation; the smallness of the pulse, and the entire absence of the collapsing character usually present in aortic regurgitation, showed that there was stenosis of the aortic or mitral orifice, which prevented an outflow of blood from the left ventricle sufficient in volume to produce the characteristic large and collapsing pulse; the rough systolic murmur at the aortic cartilage and the great hypertrophy of the left ventricle pointed to the existence of aortic stenosis. The great enlargement of the left ventricle, and the absence of the characteristic rough presystolic murmur leading up to a short first sound, seemed to exclude mitral stenosis which was not diagnosed in life. The apical systolic murmur, conducted to the axilla, implied mitral regurgitation, possibly secondary to dilatation of the left ventricle from the aortic lesions, or possibly due to primary disease of the mitral valve.

The pulsating jugulars and large and pulsating liver gave notice of the onset of tricuspid regurgitation. The stenosis of the tricuspid orifice was not

diagnosed. The existence of adherent pericardium was diagnosed by physical signs, rather than by severity of symptoms for which the valvular lesions seemed sufficient to account. The physical signs characteristic of this were as follows:—

Marked pulsation of the second, third, and fourth spaces to the left of the sternum. As far as one could judge, the recession appeared to be systolic in time, but the impression conveyed was rather that of a wave travelling from the base towards the apex, preceding the impulse of the apex beat.

The apex beat was also immovable in position during deep inspiration.

During the cardiac systole the left half of the epigastrium seemed to be drawn in and to recoil forcibly during diastole, at the same time the sternum seemed to be lifted by the forcible impulse of the hypertrophied right ventricle. The observation of the pulsating vein on the front of the chest, which emptied during the cardiac systole and filled during diastole, was important; this could not be due to tricuspid regurgitation which caused the systolic filling of the jugular veins. The vein, from its position, presumably emptied into the internal mammary vein, and the explanation suggested was, that the walls of the internal mammary vein were dragged apart during systole of the heart by the adherent pericardium, thus causing a suction action on its tributary veins. The autopsy showed that there was extensive adhesion between the pericardium and chest wall, which would support this theory.

The autopsy further revealed mitral stenosis and

tricuspid stenosis not suspected during life. It seems remarkable that with such extreme mitral stenosis the hypertrophy of the left ventricle should be so great, for usually in cases of simple mitral stenosis the left ventricle tends rather to atrophy. The explanation of the hypertrophy taking place may be one of the following: either, the damage to the aortic valves took place before the mitral valves were affected, or at any rate before the orifice had time to become stenosed, and then compensatory hypertrophy of the left ventricle took place; or, during the original attack of pericarditis, the heart became dilated and the adherence of the pericardium prevented it contracting down again to its normal size: thus the mitral orifice became mechanically enlarged, owing to the dilatation of the left ventricle, and the aortic regurgitation further caused hypertrophy of the left ventricle before there was time for the condition of mitral stenosis to became established.

Case L.

Case of Adherent Pericardium of old standing, complicated by severe anæmia.

CHARLES D., æt. 19.

Admitted to St. Mary's Hospital, April 15, 1893. Died April 19, 1893.

History of Illness.—For the last three months he has been very short of breath, and has occasionally

had slight attacks of hæmoptysis. For the last week his feet have been swollen.

He had rheumatic fever six years ago, and ever since he has had attacks of pain and palpitation in the præcordium. He had chorea ten years ago. He is by occupation a 'cellarman,' and has lived lately in underground dark cellars.

Condition on Admission.—He is extremely pale and anæmic; lips very poor colour; conjunctivæ and mucous membranes very pale; he has a cough, and is very short of breath on exertion.

Pulse, 102. Respiration, 38. Temperature, 99·5.

Cardio-vascular System.—Pulse, 102, regular in force and frequency; wave is of fair size, rather sudden and ill-sustained, the artery easily compressible. The veins in the neck are pulsating and are slightly distended.

Heart.—Apex beat visible in fifth space, ½ inch outside nipple line.

There is systolic retraction of the fifth space.

The apex beat on palpation is somewhat diffuse, but forcible and thrusting. It does not change its position during deep inspiration.

The lower end of the sternum is tilted forwards by the forcible impulse of the right ventricle.

The *area of cardiac dulness* extends upwards to fourth rib; inwards ½ inch beyond right border of sternum; outwards ¾ inch beyond vertical nipple line.

On *auscultation*, there is a loud systolic murmur at the apex, conducted into the axilla; also a rumbling presystolic murmur; the first and second sounds are audible, but are weak.

At the pulmonary cartilage and down the left edge of the sternum a soft diastolic murmur is heard; this is not heard so well at the aortic cartilage, where a weak second sound and a faint diastolic murmur are audible; the pulmonary second sound is accentuated.

The liver is much enlarged, and pulsates; it extends down to 1 inch above umbilicus. There is no œdema of the legs or the abdominal walls, but the feet are slightly œdematous.

Lungs.—Normal resonance on percussion; no adventitious sounds.

April 18th.—He is very much worse, and in a state of extreme collapse; he had a very bad night, and was sick, and became suddenly much collapsed; he lies on his left side, legs drawn up, face pale and anxious with a pinched expression; there is a clammy cold sweat all over the body; the pulse is 160 of a running character; respiration, 56, very hurried; dyspnœa intense. Temperature, 96.

Heart.—Dulness extends two inches beyond right margin of sternum, and upwards to third space; the apex beat is very diffuse and irregular.

On *auscultation* the sounds are so rapid and tumultuous that they cannot be analysed.

Liver extends down to 1 inch below umbilicus.

He is extremely restless, and in such great distress that it is difficult to examine him satisfactorily.

The condition of collapse and restlessness and dyspnœa was maintained till his death, which took place at 3 p.m. the same day.

Post-mortem Examination.

The pericardium is universally adherent to the heart by firm old and fibrous adhesions; it is also adherent to the chest wall.

The heart is much enlarged; weight, 28 ozs.

All its cavities are greatly dilated. The right ventricle is considerably hypertrophied, its walls being much thickened; it is also much dilated; the tricuspid orifice admits four fingers; the valves are normal.

The left ventricle is also hypertrophied and dilated. The mitral orifice admits three finger-tips; the aortic flap of the mitral valve has numerous vegetations on its upper surface. On the chordæ tendineæ of the anterior papillary muscle is a polypus-like projection, consisting of a calcified vegetation.

The left auricle is much dilated.

The aortic valves are incompetent, the thin edge and lunula being replaced by irregular thickened vegetations.

Lungs.—Congested and œdematous: otherwise normal.

Spleen.—Enlarged and firm; it presents traces of old infarcts.

Liver.—Enormously enlarged; typical 'nutmeg' on section.

Kidneys are hard and firm; some fibroid change: otherwise normal.

Commentary.

On admission, the most striking features were the intense anæmia of the patient—due, doubtless, to a large extent, to the fact that he worked all day in a wine-cellar underground, where there was no daylight. The dyspnœa, also, was very marked. The pulsating jugulars and enlarged and pulsating liver, on admission, showed that there was dilatation of the right ventricle and tricuspid regurgitation; the forcible epigastric pulsation and lifting of the sternum showed that there was also considerable hypertrophy of the right ventricle. That the left ventricle was also hypertrophied was evidenced by the forcible thrusting character of the apex beat. Its position was ½ inch outside the nipple line, which would be accounted for by the enlargement of the right ventricle pushing it over; but its position in the fifth space was higher up than would be expected, considering the amount of cardiac hypertrophy. There was aortic incompetence, as evidenced by the presence of diastolic murmur; but that the regurgitation was not extensive was probable from the character of the pulse, which was not markedly collapsing, and from the presence of a second sound as well as the diastolic murmur audible in the second right intercostal space. There was no marked carotid throb. What, then, had caused the onset of the symptoms of cardiac failure? Probably not the aortic incompetence, which was not severe; nor the mitral regurgitation. Tho anæmic condition of the patient was doubtless a contributory

cause, but, on examination, the systolic retraction of the fifth space, and the absolute fixation of the apex beat in a deep inspiration, seemed to point to the presence of adherent pericardium. Further, the position of the apex beat in the fifth space, when one would have expected to find it in the sixth, judging from the amount of cardiac hypertrophy, was in favour of this diagnosis. That the symptoms were so severe and out of proportion to the physical signs would further have borne out this diagnosis; but the anæmia had also to be taken into consideration. The diagnosis of adherent pericardium was made during life from the physical signs above mentioned, and from the consideration that the right ventricle had given way without there being adequate valvular lesions to account for its break-down, so that the further factor of adherent pericardium had to be brought in.

The autopsy proved the diagnosis to be correct. The intensity of the dyspnœa and the rapid death, when once signs of cardiac failure had shown themselves, were doubtless due in great measure to the anæmia.

Case M.

Case of Adherent Pericardium with Mitral Stenosis.

A. S., æt. 14.

Admitted to St. Mary's Hospital, November, 1892. Died, February 21, 1893.

Was admitted for œdema of legs and swelling of abdomen.

History.—He was in the hospital in January, and again in July, 1892, with œdema of the legs, and the diagnosis then made was mitral regurgitation and stenosis. He recovered sufficiently to go home, but has never been well since, and has had swelling of legs after walking. He had previously had two attacks of rheumatic fever, dates uncertain.

Condition on Admission.—Pale-faced boy with anxious expression; lips of poor colour; is rather thin; has orthopnœa; lower extremities œdematous. Pulse, 118. Respiration, 57. Temperature, 98·5. There is pulsation of the jugular veins in the neck.

Cardio-vascular System.—Pulse, 118, regular in force and frequency; wave small and ill-sustained: artery small, easily compressible.

Heart.—Apex beat in sixth space, just outside the vertical nipple line; impulse diffuse and very feeble.

There is marked epigastric pulsation; the lower end of the sternum is tilted up by the forcible systolic contraction of the right ventricle.

There is systolic reaction of the third, fourth, and fifth intercostal spaces to the left of the sternum.

The *area of cardiac dulness* extends upwards to the third intercostal space; inwards to about ¾ inch to the right of the sternum; outwards to 1 f. b. outside the nipple line.

On *auscultation*, at the apex a systolic murmur is heard which is conducted to the axilla, also a loud rough presystolic murmur, leading up to a short sharp first sound, which is audible as well as the systolic murmur.

To the left of the sternal notch over the tricuspid area a loud blowing systolic murmur is heard.

The pulmonary second sound is accentuated; the aortic second sound is weak; there are no murmurs at the base.

Lungs.—Respiration, 56 per minute; on percussion, the resonance is good, except at the bases posteriorly, where it is much impaired; over the impaired area coarse moist sounds are heard, especially at the end of deep inspiration.

The abdomen is distended, the walls are œdematous. There are signs of fluid in the peritoneal cavity.

The liver is much enlarged, and extends down to the level of the umbilicus; it pulsates.

Urine.—Sp. gr. 1026; pale; acid reaction; no albumen; thick deposit of urates.

The further history of the case is that of a progressive increase in severity of all the symptoms, except for a slight improvement which followed his admission. In spite of rest and treatment—which included purging and leeching, and removal of fluid by Southey's tubes, and the free use of digitalis and strychnia—he got steadily worse, eventually gangrene of the right leg set in, due to thrombosis of the right iliac vein. He died on February 21, 1893. The œdema was then universal and extreme.

Post-mortem Examination.

The pericardium was universally adherent to the heart, and partially to the chest wall, by old tough adhesions.

The heart was considerably enlarged.

The left auricle was much dilated and also hypertrophied.

The left ventricle was enlarged, but was not much hypertrophied.

The mitral orifice admitted one finger; the valves were opaque, thickened and rigid, and adherent at their margins.

The right ventricle was considerably dilated and hypertrophied.

The tricuspid orifice admitted four fingers. The valves were somewhat thickened at edges by old vegetations.

Lungs and Pleura.—There were no adhesions between the lungs and chest wall.

The lungs were œdematous at the bases, but were otherwise normal.

Abdomen.—There were about 20 ozs. of fluid in the peritoneal cavity.

The liver was very much enlarged and was 'nutmeg' on section.

Kidneys.—Capsule strips readily; substance rather firm; no macroscopic changes.

The vena cava inferior and common iliac veins were full of clot, which extended downwards into the right femoral vein for some distance.

Commentary.

The œdema of the legs, the pulsating jugulars, and the enlarged and pulsating liver indicated tricuspid incompetence and obstruction to the venous return. The symptoms were thus very severe, and were those of right ventricle failure. The forcible epigastric pulsation showed that the right ventricle was considerably hypertrophied as well as dilated. The rough vibratory character of the presystolic murmur at the apex, leading up to the short sharp first sound, pointed to mitral stenosis, and the systolic apical murmur to mitral incompetence.

The question to be decided was, whether the mitral lesions were sufficient in themselves to account for the break-down of the right ventricle, by causing obstruction to the entry of blood to the left auricle, and consequently backward pressure through the lungs, thus throwing more work on the right ventricle than it could perform, or whether there was some other factor as well.

There was no cyanosis, no hæmoptysis, and no evidence of severe pulmonary congestion, such as one would expect if the mitral lesions were alone responsible.

Further, the right ventricle was obviously much hypertrophied, more so than appeared necessary to compensate for the valvular lesions; there was also a history that compensation had already broken down on two previous occasions and had been with difficulty restored, and the boy had never been free from

œdema since, except while in bed. On this occasion the break-down of the right ventricle was complete, and rest and treatment failed to improve its condition. One would not expect the stenosis of the mitral orifice to be so advanced in so young a subject as to give rise to permanent failure of compensation, nor, as far as one could judge from the physical signs, was the mitral obstruction very severe.

Adherent pericardium was therefore suspected as an important contributory cause of the excessive hypertrophy of the right ventricle and also of its permanent break-down. There was also some enlargement of the left ventricle, as was shown by the presence of the apex beat in the sixth space. This could not be accounted for by the mitral obstruction, which would rather tend to cause atrophy than hypertrophy of the heart; nor was the mitral regurgitation, slight in amount, as far as one could judge, sufficient to cause it; hence adherent pericardium was thought to be responsible also for the enlargement of the left ventricle.

Among the physical signs, the systolic recession of the second, third, and fourth intercostal spaces supported the diagnosis of adherent pericardium, which was made during life from the considerations just stated.

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